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PRELIMINARY ASSESSMENT OF THE ENVIRONMENTAL PROBLEMS ASSOCIATED WITH VINYL CHLORIDE AND POLYVINYL CHLORIDE

ENVIRONMENTAL PROTECTION AGENCY

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The Report is organized into an Executive Summary, three Sections, and Appendices. The first Section discusses the nature and magnitude of the problems associated with vinyl chloride and polyvinyl chloride activities. The second Section discusses previous and planned activities within the Federal Government of particular significance and the role of industry. The Report concludes with a Section setting forth the specific recommendations of the Task Force. The Appendices present a considerable body of original data developed by the Task Force with the assistance of a large number of EPA specialists.

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PRELIMINARY ASSESSMENT OF THE ENVIRONMENTAL PROBLEMS
ASSOCIATED WITH
VINYL CHLORIDE AND POLYVINYL CHLORIDE

A Report on the Activities & Findings of the

Vinyl Chloride Task Force

Compiled by the Office of Toxic Substances
Environmental Protection Agency
Washington, DC
September 1974

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PREFACE

This Report summarizes the activities and findings of the Task Force established by the Administrator on February 14, 1974, to assess the character and extent of the problems associated with the production, distribution, use, and disposal of vinyl chloride and polyvinyl chloride. The discussion and conclusions presented in the Report should be considered preliminary since the Task Force has only scratched the surface of a complicated subject and analyses within the Agency are continuing. The Report is not a statement of Agency policy even though many of the recommendations are already being implemented.

During the lifetime of the Task Force the Agency took several regulatory steps concerning vinyl chloride. These were directed to banning pesticidal sprays containing vinyl chloride as a propellant and requesting information from industry pursuant to Section 114 of the Clean Air Act concerning vinyl chloride air emissions and related control technologies. Since these activities have been or are being documented in detail in other reports, they are mentioned only briefly in this Report.

Similarly, in recent months the Occupational Safety and Health Administration, the Food and Drug Administration, and the Consumer Product Safety Commission took a series of regulatory actions directed to vinyl chloride. While recognizing many of the problems facing other Government agencies and the implications for EPA of their regulatory steps, the Report dwells primarily on those activities of direct responsibility to EPA, and problems such as worker exposure to chemicals, use of polyvinyl chloride in food packaging, and consumer products containing vinyl chloride have not been a major concern.

The Report is organized into an Executive Summary, three Sections, and Appendices. The first Section discusses the nature and magnitude of the problems associated with vinyl chloride and polyvinyl chloride activities. The second Section discusses previous and planned activities within the Federal Government of particular significance and the role of industry. The Report concludes with a Section setting forth the specific recommendations of the Task Force. The Appendices present a considerable body of original data developed by the Task Force with the assistance of a large number of EPA specialists.

TABLE OF CONTENTS

Page

PREFACE i

EXECUTIVE SUMMARY 1

- Background 1
- EPA Concerns 2
- Further Steps 3

CHARACTER AND SCOPE OF PROBLEMS BEYOND THE WORKPLACE 4

- Emergence of the VC Problem 4
- Exposure to VC of Special Importance to EPA 5
  - Pesticidal Sprays
  - Discharges from VC/PVC Plants: Materials Balance and Monitoring Data
  - Transportation Accidents
  - Unreacted Monomer Entrapped in PVC Products
- Health Effects of Vinyl Chloride 8
- Persistence of VC 10
- Ecological Effects of VC 10
- Other Chemicals of Concern in PVC Activities 11
- Size and Character of the PVC Industry 12
- Lessons Learned from VC/PVC Experiences Relevant to Other Chemical Problems 13

INTERESTS AND ACTIVITIES OF GOVERNMENT AGENCIES AND INDUSTRY 16

- EPA Regulatory Authorities 16
  - Pesticide Registration
  - Air Pollution
  - Water Pollution
  - Solid Waste Disposal

Page

Ocean Dumping
Drinking Water
Proposed Toxic Substances Control Act
Supporting Research Activities

Regulatory Interests of Other Agencies 19

Aerosol Sprays
Worker Protection
Unreacted VC Monomer in PVC Products
Transportation Handling
Related Research Activities

Role of Industry 22

Reducing Discharges of VC and Other Toxic Chemicals
Medical Surveillance
Fenceline Monitoring for Chemical Discharges
Toxicological Testing of VC
Testing for Persistence and Environmental Fate and Effects of VC and PVC
Testing for Levels of Unreacted Monomer in PVC Resins and PVC Products

RECOMMENDATIONS 26

Regulatory and Directly Supportive Actions by EPA 26

Steps by Industry 29

APPENDICES

APPENDIX I - SELECTED ECONOMIC CONSIDERATIONS

APPENDIX II - PRODUCERS OF VC AND PVC

APPENDIX III - THE MATERIALS BALANCE AT VC AND PVC FACILITIES

APPENDIX IV - INTERIM METHOD FOR SAMPLING AND ANALYSIS OF VC IN WASTE WATER EFFLUENTS AND AIR EMISSIONS

APPENDIX V - SUMMARY OF REGIONAL ACTIVITIES

APPENDIX VI- PERSISTENCE OF VINYL CHLORIDE

APPENDIX VII- HEALTH EFFECTS OF VINYL CHLORIDE

APPENDIX VIII- DISPOSAL OF PRODUCTS CONTAINING POLYVINYL CHLORIDE

APPENDIX IX - ACTIVITIES OF TASK FORCE

EXECUTIVE SUMMARY

Background

EPA's recent concern over vinyl chloride (VC) and polyvinyl chloride (PVC) was triggered by reports in January 1974 of (a) deaths of four PVC workers believed to be attributable to exposure to VC, and (b) an estimated material loss of VC and PVC of six percent during the PVC polymerization process. The investigations of the Task Force confirm that in the United States substantial amounts of VC--probably exceeding 200 million pounds annually -- and large quantities of PVC -- probably exceeding 50 million pounds -- are being discharged into the environment during the PVC production process. Meanwhile, additional epidemiological and toxicological evidence supports the linkage between worker deaths and VC exposure.

The VC/PVC industry has experienced rapid growth at home and abroad in recent years with PVC products currently permeating our entire economy. This multi-billion dollar industry involves not only several dozen large manufacturers of VC and PVC resin but also thousands of fabricators producing a variety of products based on PVC and probably employing more than 300,000 workers. There are readily available substitutes for PVC in some of these products; more expensive substitutes for others; and no substitutes for still others. A principal constraint on near-term production increases has been availability of ethylene, a petroleum derivative. A new constraint is the uncertainty over Governmental regulatory actions affecting VC/PVC activities.

VC has induced angiosarcoma of the liver in rats and mice exposed to concentrations of 50 ppm at intervals simulating occupational exposure, and this same rare and fatal tumor has been identified in at least 15 former workers in U. S. PVC facilities. Cancers at other sites, non-malignant liver disease, and a unique occupational disease -- acroosteolysis -- have also been attributed to VC exposure. There is no direct evidence that VC contributes to adverse health effects at the lower levels of exposure encountered outside the workplace; however, two recently reported cases of angiosarcoma of the liver in non-workers who had lived near PVC fabrication plants suggest the possibility of such a correlation. At the same time there has been little effort to search for adverse effects at these lower levels. Since carcinogens are generally considered not to have a "no effects" threshold level, there should be concern with possible risks to health at even the lowest levels of exposure to VC which are encountered in the ambient air, and possibly in water.

Preliminary monitoring results at seven industrial complexes involving 10 PVC resin and 2 VC plants indicate that the levels of VC in the ambient air near the plants fluctuate sharply, apparently due to the periodic opening of reactor kettles in the PVC plants, accidental plant discharges, variations in the production process, and meteorological conditions. While almost all of the samples collected contained detectable levels of VC, more than 90 percent of the instantaneous observations and 97 percent of the 24-hour samples showed amounts less than 1 ppm. At several of the chemical complexes, a number of individual air samples showed more than 1 ppm. At one PVC plant a high of 33 ppm was obtained at one site; however, repeated sampling indicated that this level was an unusually high excursion with the average at this site less than 1 ppm. Most of the air sampling was done within one-half mile of the property lines of the plants. In one case a level of 3.4 ppm was recorded at a distance of three miles from the plant, but the average of several readings at this site was approximately 0.5 ppm.

Water effluents were also monitored, and levels varied considerably depending on the in-plant handling of wastes and treatment of wastewater. The highest level for wastewater leaving the plant site was 20 ppm. More typically, levels of 2 to 3 ppm were found. Not unexpectedly, the levels of VC entrapped in sludge and other solid wastes from the reactor kettles ranged from 100 ppm to, in one case, 3,000 ppm.

EPA Concerns

The principal near-term issues facing the Agency include (a) an assessment of the risks associated with exposure to VC, with such an assessment hampered by inadequate data concerning health effects at the levels likely to be encountered in the environment and only very preliminary monitoring results, (b) the costs to industry to reduce the levels of VC entering the environment, keeping in mind that much of the cost will be passed on to society as a whole, and (c) regulatory and related steps which will contribute to reducing the risks at an appropriate cost. Two closely related areas of interest are (a) the amount of Agency resources to be directed to VC/PVC in the months and years ahead, and (b) lessons that have been learned in addressing VC/PVC that can save time and resources in addressing other chemicals.

VC air emissions from PVC polymerization plants -- and to a lesser extent from VC plants and possibly PVC fabrication plants -- are the most immediate concern to the Agency. The earlier problems related to the use of VC as a propellant in pesticidal sprays have been largely resolved with the banning of such sprays. Other current concerns related to VC include the Agency's capability to provide sound advice in the event of transportation accidents involving the release of VC; the fate and effects, if any, of VC trapped in water effluents, and particularly VC that might thereby appear in drinking water; and the migration of unreacted VC from PVC products into drinking water or into the ambient air.

There are of course a host of other chemicals involved in VC/PVC activities that should be of concern. Impurities in the VC, PVC as inhaled or ingested particulate, additives introduced to alter the properties of PVC, copolymers used in conjunction with PVC, and chlorinated hydrocarbon wastes from VC production all need further investigation. The by-products associated with PVC incineration and the leaching of additives, and particularly plasticizers and toxic metals, from PVC products which come into contact with the aquatic environment need additional study.

Further Steps

Development of an air emission standard for VC is currently underway, with additional monitoring activities being planned in the immediate future to improve the basis for the standard. While additional epidemiological and toxicological data are slowly becoming available, it is unlikely that there will be a good technical basis within the next few months or perhaps even within several years for establishing an appropriate ambient level to protect public health. Therefore, a performance standard may be far easier to develop and implement than a standard based upon achieving a specific air quality level. Initial estimates indicate that available control technology when installed can reduce VC emissions by about 75 percent from PVC resin plants and 90 percent from VC plants with a concomitant increase in the cost of PVC of about four percent. Currently available information indicates that such reductions should result in ambient air levels which present no established risk to health or welfare. Meanwhile, close liaison with the Department of Labor is important to insure that the regulatory approach to protect the worker is compatible with EPA regulatory activities.

The Agency should monitor for VC in drinking water supplies. Monitoring is also needed in the ambient air -- indoors and outdoors -- distant from chemical complexes to determine whether concentrations of PVC products release quantities of unreacted VC that should be of concern. Further refinement of monitoring methodologies is needed in support of these efforts. The limited toxicological and epidemiological studies that are planned should be fully supported.

The Agency should continue its leadership role in bringing together other Agencies to exchange views on regulatory actions, supporting activities, and research projects related to VC and PVC. Industry should be strongly encouraged to accelerate its efforts to reduce VC discharges and its very limited research, testing, and monitoring activities to clarify further the problems associated with VC and PVC.

Finally, we have been alerted in a rather dramatic fashion to the need for greater attention to an important segment of our industrial base which will surely continue to expand in the years ahead. Many of the considerations and uncertainties that have punctuated the VC/PVC deliberations undoubtedly characterize a far broader swath of concerns over high volume industrial chemicals in general, and plastics in particular. Hopefully, we can extrapolate from current experiences with VC and PVC in identifying problems with other potentially important commercial chemicals early in their embryonic stage and thus minimize health and environmental hazards and also the economic dislocations attendant to corrective actions. In this regard the Agency should continue its efforts to seek early enactment of the Toxic Substances Control Act which can provide a much needed broader basis for addressing these types of problems.

CHARACTER AND SCOPE OF PROBLEMS BEYOND THE WORKPLACE

Emergence of the VC Problem

On January 22, 1974, the B. F. Goodrich Company, the largest U. S. producer of PVC resin, notified the National Institute of Occupational Safety and Health (NIOSH) that four workers from its PVC polymerization plant in Louisville, Kentucky, apparently had died from a rare cancer, angiosarcoma of the liver. All four workers had been closely associated for many years with the production of PVC resins. The rarity of the tumor and the clustering of deaths at a single plant raised suspicions that an occupational disease related to VC* exposure had been found. Since that time, 10 additional cases of this tumor, which developed in U.S. PVC polymerization workers since 1961, have been confirmed. This tumor has also been reported in seven workers at European polymerization plants, one worker at a U.S. PVC fabrication plant, two workers at European fabrication plants, one worker at a European VC plant, and two residents in the general population near U.S. fabrication plants.

Concurrently, toxicological data from animal studies became available which further strengthened the suspicion of VC as the etiological agent in the formation of the liver cancer. A broad spectrum of cancers was reported by Professor Cesare Maltoni of Italy in different animal species at various exposure levels. His inhalation studies of rats exposed to 50 ppm at repeated intervals approximating occupational exposures have produced angiosarcomas of the liver and abdomen as well as tumors of the kidney and skin. In mice exposed to VC the same tumors have been observed, with the addition of lung tumors. Animal studies sponsored by U. S. industry have confirmed Maltoni's observations at 50 ppm. Recent epidemiological studies also suggest the possibility of multiple cancers attributable to VC exposure.

Meanwhile, statements by industry and Government officials indicated that the material loss to the environment during the PVC polymerization process may be about six percent, with more than 75 percent of the losses being VC air emissions. Also, it soon came to light that VC was being used as a propellant in pesticidal sprays, and the EPA Regional Offices were becoming more aware of railroad accidents involving VC tank cars.

Until this series of events the Agency had not been particularly concerned with VC as an urgent problem. PVC plants had been on the list of industries to be examined as candidates for new source performance standards to limit air emissions. Also, limited studies of PVC disposal were underway, and VC and PVC resin manufacturing activities have been addressed in the Effluent Guidelines promulgated under the Federal Water Pollution Control Act. However, only since January have VC/PVC activities been elevated to the level of priority Agency attention.

** Vinyl chloride (CH_2CHCl) is a colorless, faintly sweet smelling gas at room temperature. As a gas it is readily flammable and explosive but is usually handled industrially as a liquid under pressure. Polyvinyl chloride resin is a fine powder which is produced by polymerizing vinyl chloride. The resin is the base ingredient for a wide variety of plastics.*

Exposure to VC of Special Importance to EPA

Pesticidal Sprays

Spurred by the active interest of a consumer protection organization, the Health Research Group, one of the earliest EPA concerns this year was the use of VC as a propellant in a large number of pesticidal sprays. After the searching of EPA pesticide registration files more than 50 different sprays containing VC -- including a large number used indoors -- were identified. It has been estimated that in addition to the cans in the possession of consumers, up to 100,000 cans were in the channels of trade.

Preliminary tests at EPA research facilities showed that a 30 second release of an aerosol containing VC could result in a concentration as high as 400 ppm in the air. Related tests showed that in closed rooms VC will persist for many hours, and even when diluted by ventilation, will probably result in some VC exposure within the household for at least several hours. (See Appendix VI).

Discharges from VC/PVC Plants: Materials Balance and Monitoring Data

Industrial reports and analytical studies (see Appendix III) confirm that generally the material loss during the PVC polymerization process ranges from 4.5 to 7.5 percent. During recent months industry has taken a variety of steps to reduce the losses, and these losses may be now declining. The losses will vary with the type of process, the age of the plant, the level of technology that is employed, and manufacturing practices. However there is no doubt that in the United States substantial amounts of VC -- probably exceeding 200 million pounds annually -- and large quantities of PVC -- probably exceeding 50 million pounds -- are being discharged into the environment during the PVC polymerization process. Most of the VC escapes directly into the atmosphere, with lesser amounts dissolved in water effluent streams and entrapped in sludge and solid wastes. PVC losses occur as particulate in air emissions, suspended solids in water effluents, and components of solid wastes.

A principal area of VC leakage is associated with the operation of the polymerization kettles, including losses when they are opened or when they are recharged or sampled. Other losses occur during the transfer of VC from tank cars to storage, during the PVC drying process, and from leaks at a variety of valves, flanges, and pump seals throughout the process. Polymer losses are similarly distributed among a variety of activities including dust collector losses, disposal of oversize particles, and sampling losses. In this regard two aspects are particularly significant: there are a variety of PVC processes with differing problems and control possibilities, and in every case the number of potential leakage points is very large.

Losses at VC plants occur during the loading process, as the result of venting of gases, from leaks in pumps, and at other points. These losses are considerably less than one percent but still may be environmentally significant. As in the case of PVC polymerization activities, current industrial efforts should reduce these losses. No mass balance data are available concerning losses at PVC compounding and fabrication plants. However, the only source of VC at these facilities is the unreacted monomer in the PVC resin which suggests that environmental discharges are less than at the VC and PVC polymerization plants.

To obtain more direct evidence on VC discharges from VC/PVC activities, ambient air, waste water effluents, and solid wastes from seven chemical complexes were monitored during May. The 2 VC and 10 PVC production facilities located at these seven complexes are listed in the table below:

Vinyl Chloride and Polyvinyl Chloride Manufacturing Complexes
Monitored by EPA Regional Offices

Louisville, Ky.	PVC Plant	The B. F. Goodrich Co. B. F. Goodrich Chemical Co.
Leominster, Mass.	PVC Plant	Borden, Inc. Borden Chemical Division
Plaquemine, La.	PVC Plant	The Goodyear Tire & Rubber Co. Chemical Division
	VC Plant	Dow Chemical, U.S.A.
Long Beach, Calif.	PVC Plant	The B. F. Goodrich Co. B. F. Goodrich Chemical Co.
	PVC Plant	American Chemical Corporation
	VC Plant	American Chemical Corporation
Painesville, Ohio	PVC Plant	Uniroyal, Inc. Uniroyal Chemical Division
	PVC Plant	Robintech, Inc.
Delaware City, Del.	PVC Plant	Stauffer Chemical Co. Plastics Division
	PVC Plant	Diamond Shamrock Corporation Diamond Shamrock Chem. Co.
Flemington, N. J.	PVC Plant	Tenneco, Inc. Tenneco Chemicals, Inc.

The preliminary monitoring results indicate that the levels of VC in the ambient air near plants fluctuate sharply, apparently due to the periodic opening of polyvinyl chloride reactor kettles, accidental plant discharges, variations in the production process, and meteorological conditions. A summary of the monitoring results at each complex can be found in Appendix V. The monitoring method developed for this activity, and subsequently refined, is presented in Appendix IV. Since the samples were limited in number, time, and duration, additional monitoring is in order to obtain a more definitive assessment of VC discharges.

Most of the air sampling was done up to three miles beyond the property lines of the plants although in two regions the EPA sampling teams were able to conduct part of the study within the plant property. Almost all air samples contained detectable levels of VC. However, more than 90 percent of the instantaneous samples and 97 percent of the 24-hour samples showed less than 1 ppm. In one case a level of 3.4 ppm was recorded at a distance of three miles from the plant. The average of several readings at this site was about 0.5 ppm. A high value of 33 ppm was observed around one complex at a distance of 0.3 miles from the fence-line although the average at this site was less than 1 ppm.

The levels of VC in water effluents varied considerably depending on the in-plant handling of wastes and treatment of wastewater. The highest level for wastewater leaving the plant site was 20 ppm. More typically, levels of 2 to 3 ppm were found. Not unexpectedly, the levels of VC entrapped in sludge and other solid wastes from the reactor kettles ranged from 100 ppm to 3,000 ppm.

Transportation Accidents

It is estimated that more than two-thirds of the produced VC is transported from the production site to another location, frequently located hundreds of miles away. More than 95 percent of the shipments travel by rail tank car, with a small amount being shipped by water. During the past three years there have been 16 reported rail accidents involving VC tank cars. The immediate concern has been prevention of fire and explosion and only recently has attention been directed to the long-term effects, if any, that might be associated with a one-time massive exposure to VC. 1/

Unreacted Monomer Entrapped in PVC Products

Industry has reported to EPA that PVC resin contains in very unusual cases as high as 8,000 ppm of unreacted VC monomer although the levels are usually between 50 and 1,000 ppm. Presumably these levels are substantially reduced as the resin is processed further, with much lower levels (e.g. 5 to 20 ppm) present in finished products containing PVC.

Nevertheless, the eventual fate of the unreacted monomer in PVC products is of concern. Conceivably, it could be contributing to a general environmental background level of VC, and detectable levels of VC may be present where there is a heavy concentration of products containing PVC, and particularly new products.

In view of EPA's responsibilities concerning drinking water, a problem of special significance is the possible migration of unreacted monomer into the water from PVC pipe or liners used in drinking water systems. At present little is known about such migration.

Health Effects of Vinyl Chloride

There is no direct evidence about the health effects on man of VC at the levels of exposure that have been or are likely to be encountered outside the workplace; however, the two recently reported non-occupational cases of angiosarcoma of the liver in neighborhoods near PVC fabrication plants suggest the possibility that there may be a correlation between the incidence of angiosarcoma and low levels of exposure. There are also recent reports of angiosarcoma among workers at fabrication and VC plants who presumably were exposed to relatively low levels of VC. At the same time epidemiological and toxicological studies have clearly linked VC to angiosarcoma of the liver and other adverse effects at the higher levels of exposure that have been encountered in the workplace. Given the previous lack of effort to search for effects at low doses, it seems prudent to assume that there probably is not a no-effects threshold for VC, and there should be concern about the possible health effects at any level of exposure.

Extrapolation of effects from animals to man, from intermittent to sustained or peak exposures, and from high to low dose levels is fraught with uncertainty. Nevertheless, such extrapolations can be useful in helping set the basis for the necessarily subjective judgements that must be made as to health risks. With regard to the toxicological data, studies are currently underway at the National Cancer Institute and within EPA using statistical methods to extrapolate from the effects at 50 ppm to likely effects at 1 ppm and lower. Such statistical techniques must be viewed with caution but can be helpful in providing a sense of perspective in assessing risk. The relative sensitivites of rats, mice, and men to VC are not known, nor is there good information on the relative sensitivities within a human population. Another difficulty is interjected in attempting to deal with the extrapolation from worker exposure (i.e. 40 hours per week) to neighborhood exposure (i.e. up to 168 hours per week), even assuming that time weighted averages are the determinant in ambient air rather than peak levels.

The risks, if any, associated with ingestion of VC, via drinking water, food, or other routes are totally unknown. It is prudent to assume that ingestion is no less worrisome than inhalation although the likelihood of sustained ingestion at even low levels seems remote.

Summarized below are some of the most important known health effects of VC. A more detailed presentation of health data is set forth in Appendix VII, recognizing that additional information is continuously becoming available. Ongoing Agency studies are taking this information into account.

Anesthetic Effects of Acute Exposures: These effects have occurred in PVC workers exposed to high concentrations of VC as the result of accidents or inadequately ventilated reactor vessels. A dizziness, nausea, and loss of consciousness occur which are reversible upon exposure to fresh air. Human volunteers have reacted in a similar way to high levels of VC inhaled for brief periods. These subjects reported feeling dizzy or inebriated, experienced loss of certain reflexes, and encountered feelings of impending unconsciousness. The effects were proportional to concentrations and duration of exposure.

Acroosteolysis: A small proportion (from 1 to 3 percent) of workers involved in manual cleaning of PVC reactor vessels have experienced a combination of symptoms known as acroosteolysis. This is characterized by a soreness and thickening of the skin at the fingertips, a gradual dissolution of bone calcium at fingertips and toes, skin sores, and frequently heightened sensitivity of the hands to cold (blanching of the skin and pain). These symptoms were first described in 1967, and apparently occur only after several years of high levels of exposure.

Liver Function Abnormalities: Changes in blood chemistry attributed to alterations in liver function have been observed in PVC workers whose 8-hour exposures to VC averaged 300 ppm. At levels below 300 ppm, the noted functional changes were minimal, suggesting a dose-response relationship with respect to liver function. There has been no overt liver disease noted. From Europe, there are reports that liver damage was found in Russian workers, and disease of the liver, skin, and other organs has been discovered in workers in Rumania and France. Other chemicals are invariably present in the occupational setting, and may have interfered with the effect often attributed to VC alone.

Liver Angiosarcoma: A fourth effect related to occupational exposure is angiosarcoma of the liver, a rare form of liver cancer which is a progressive and invariably fatal disease. It has recently been reported in 15 workers in PVC facilities in the United States. The exposure time of these workers in the factory setting has ranged from 11 to 30 years. Many of these workers were engaged in cleaning polymerization reactor vessels in an area where exposure levels were the highest.

The etiological role of VC in the induction of liver angiosarcoma is not certain because of possible confounding factors. The occupational exposure of the workers to other chemicals precludes identifying this substance as the sole cause for cancer. However, taking into account the effect of VC on animals in the absence of other chemicals, the implication of VC as the possible etiological agent is very strong, and unless another carcinogenic agent is identified, VC must clearly remain the prime suspect.

The reported cases of angiosarcoma of the liver have in the past been extraordinarily rare. For example, in the Third National Cancer Survey (1969-1971), a population of 21 million persons residing in nine geographic areas was sampled for incidence and type of cancer. In this period, only eight cases of liver angiosarcoma had been newly diagnosed among that ten percent of the U.S. population.

Controlled Exposures of Animals to VC: On laboratory mammals VC concentrations of 5 to 20 percent have narcotic effects which are not unlike the human anesthetic effects noted above. The acroosteolysis syndrome has never been produced in animals, although disturbances in peripheral blood circulation, abnormal cartilage formation in the toes, and abnormal skin lesions have occurred in rats. Liver enlargement in rats has been seen with inhalation exposure as low as 100 ppm for 6 months, but little is known about frank liver disease caused by VC exposure.

Among the several animal inhalation experiments employing repeated exposures of over six months duration, two have disclosed liver angiosarcoma at 50 ppm and higher in mice and rats. It remains to be determined whether the liver angiosarcoma observed in animals are precisely the same as those reported for occupationally exposed workers and for nonworkers. Nevertheless, the similarities observed between the animals and man strongly suggest that the animal carcinogenic reaction is like that reported for PVC workers.

Persistence of VC

As discussed in Appendix VI, very preliminary investigations at EPA laboratories suggest that in the ambient air near the emission source VC can be considered as a stable pollutant whereas VC rather quickly escapes from agitated or aerated water.

The available results indicate a rate of reaction of about 8 to 10 percent per hour for VC in air. The direct and indirect reaction products identified include ozone, nitrogen dioxide, carbon monoxide, formaldehyde, formic acid, and formyl chloride. High eye irritation levels found with human exposure levels are consistent with these products. Although VC would disappear significantly over longer travel distances, the conversions anticipated within a few miles downwind of VC emission sources indicate that VC can be considered a stable pollutant near emission sources. The usual meterological dispersion equations for gases could be applied to approximate concentrations. Because of strong inversions at night during the fall and winter period, buildup of VC near emission sources might be of particular concern during such periods.

Ecological Effects of VC

As indicated above, preliminary studies concerning the volatility of VC from aqueous solutions as well as analyses of hydrolysis and photolysis suggest that the impact of VC in the aquatic environment may not be significant. However, if there is a continued input of VC into water, it is possible that a steady state concentration of VC could be reached. Bioaccumulation and/or biotransformation might then become of considerable concern.

Potential terrestrial ecosystem effects, as well as transport pathways from source to plant or animal receptors, of VC are unknown. However, the suspected volatility of VC may result in a large dilution factor which might reduce the toxicological or bioaccumulation hazard to an insignificant level.

Given the lack of past attention to the ecological effects of VC, some inferences drawn from the behavior of other low molecular weight chlorinated hydrocarbons may be helpful in anticipating the fate and effects of VC. In particular, 1, 1, 2 trichloroethane and 1, 2 dichloroethane comprise a portion of the waste products -- often referred to as tars -- from the production of VC and are of concern in themselves as well as possibly suggesting VC behavioral patterns. Other potentially hazardous compounds, such as hexachlorobenzene and hexachlorobutadiene, are also found in these tars. Therefore, tars are discussed below.

Other Chemicals of Concern in PVC Activities

Immediately following the January report of worker deaths related to VC exposure, questions arose as to whether other chemicals, such as vinylidene chloride, might also contribute to angiosarcoma either independently or in conjunction with VC. Little work has been done to date to clarify this concern. Also, the toxicity of PVC particulate has become a significant interest. There have been some inhalation toxicology studies conducted on PVC. However, there are no readily available data to indicate whether any substantial risk is involved at the levels of exposure that might be encountered via inhalation or ingestion of PVC outside the workplace. 2/

As pointed out in Appendix VIII, a large number of chemicals are used in PVC products as antioxidants, antistatics, colorants, fillers, plasticizers, and stabilizers, and many of them can reach man through a variety of routes. The health effects of some of these chemicals are reasonably well known; the effects of others have yet to be explored. Several of them are particularly good candidates for further investigation, e.g. cadmium, barium. However, the Task Force has not attempted to assess in any detail the known health risks nor sort out the priorities for further investigation.

A special concern of the Task Force has been disposal of products containing PVC and disposal of the by-products of PVC/VC plants. As discussed in Appendix VIII incineration of PVC produces HCl and possibly metallic vapors. As the volume of PVC and other plastic products entering municipal waste systems continues to grow, the possibility of problems resulting from incineration and land disposal -- particularly leachates -- will increase. In addition, toxic leachates from PVC used in the lining of fish tanks are known to have caused damage to aquatic organisms. 3/

The adverse effect of the tars from VC plants on aquatic organisms has been of special concern since they are known to affect at least some marine species at concentrations of 2.5 ppm. Worms and barnacles are also affected at levels below 5 ppm. Bioaccumulation of the tars appears possible through the food chain, and these materials also adhere to particles in water. However, relatively short biological half lives and rapid excretion rates may prevent serious accumulation of the tars. 4/

Size and Character of the PVC Industry

During 1973 VC production in the United States was at the 5.35 billion pound level with PVC and its copolymers at the 4.56 billion pound level. The industry has been operating for about forty years, and over the past five years has shown an annually compounded growth rate of 14 percent -- a rate of growth that is expected to taper off only moderately in the next few years. A few of the most significant characteristics of the industry are summarized below with additional details presented in Appendices I and II. 5/

PVC has become a very important polymer as evidenced by the broad dependence of nearly every branch of industrial and commercial activity upon products and components fabricated from this plastic. Markets include the apparel, building, construction, electrical, home packaging, recreation, and transportation industries. The wholesale value of the annual output of fabricated products is at least several billion dollars.

The synthesis of VC is conducted in fifteen U. S. plants, and thirty-seven plants are engaged in polymerization with almost all of these plants currently operating at or near capacity. Five new PVC resin plants are under construction and together with expansions at five others will yield an additional annual capacity of 1.378 billion pounds. Additional plants are involved in manufacturing copolymers using VC. Approximately 7500 plants are engaged in fabricating products from PVC. It is estimated that 1000 to 1500 workers are employed in monomer synthesis, an additional 5000 are engaged in PVC polymerization operations, and approximately 350,000 are associated with the 7500 PVC fabrication plants.

More than 97% of the monomer is used for the manufacture of homopolymer and copolymer resins, with the remainder utilized primarily for (a) the production of methyl chloroform, (b) additives in specialty coatings, and (c) until recently, aerosol propellants. Over 90% of the VC produced in the United States is manufactured by ethylene-based processes; the remaining 10% is synthesized by the acetylene-based process. In both processes VC is made by a continuous rather than a batch process.

PVC resins are manufactured by four polymerization processes: suspension - 79% of total; emulsion - 13%; bulk - 6%; and solution - 2%. These are all batch processes.

The number of PVC fabrication plants is at least several thousand but no complete compilation by company, location, and capacity is known to exist. For example, the B. F. Goodrich Company alone supplies finished resins and compounds to about 2200 U. S. customer plants. Some of the more important products are set forth in Appendix II.

Appendix I discusses the opportunities for PVC substitutes at comparable and at higher price. In many cases there are substitutes; in some cases there are no substitutes. At the present time worldwide shortages of ethylene and the uncertainty of regulatory actions concerning VC are creating more intensive searches for alternatives to PVC resins in a variety of applications.

Lessons Learned from VC/PVC Experiences Relevant to Other Chemical Problems

Except for continuing concern over spills and accidents, Government and industry have been rather complacent with regard to the potential environmental threat from the high volume industrial chemicals (e.g. the top fifty in terms of production levels). This complacency is in large measure attributable to the relative absence of visible and uncontrolled dangers from exposure to the chemicals during their long histories. In addition, since each of these chemicals is manufactured by a number of companies, firms may lack incentive to invest individual company resources to clarify the safety aspects of their usage. Clearly, the experience with VC -- the twenty-second leading chemical in terms of production -- underscores the problems that can result from such complacency. Despite the continuing commercial importance of these high volume chemicals, it cannot be assumed that adequate research, testing, and related safety measures will be taken by industry, and vigorous governmental leadership in this area seems essential.

The Agency's experience in addressing VC discharges from PVC plants has highlighted the need for three key types of information for decision-making and the difficulty of such decisions in the absence of adequate information:

- The levels of exposure to populations beyond the fenceline, with monitoring data being the key ingredient in estimating such levels.
- The health and environmental effects of the levels of exposure that are encountered, drawing on available epidemiological, toxicological, and ecological data from all sources.
- The feasibility -- in terms of technology, cost, and time -- of introducing controls to reduce discharges.

In all of these areas, concerted short-range efforts enhanced significantly the existing data base and provided critical inputs into the decision-making process.

Reliable techniques for sampling and analysis of VC were not available and had to be developed in a short period of time. Similarly, monitoring personnel gained their experience during the actual operations. While each chemical problem that arises will undoubtedly have unique characteristics, EPA should be able to improve its anticipatory monitoring capability by such steps as limited stockpiling of equipment (e.g. Tedlar bags), clarification of organizational and funding responsibilities, and development of immediately available contractor support services.

In the pesticides area, the use of VC in aerosol propellants has emphasized the need for greater attention to the many inert ingredients in pesticides. With regard to water pollution and to drinking water contamination, EPA must extend its emphasis beyond the traditional concerns with gross pollution effects and with toxic metals and pesticide contaminants to include a wide range of other organic chemicals. Similarly, in the area of air pollution, VC has awakened the entire environmental community to the broad problem of uncontrolled and unmonitored chemical discharges reaching neighborhoods adjacent to chemical complexes. Also, the potential problems associated with the incineration and burial of PVC products may be common to a variety of plastics.

Even at this late date all the commercial products using VC have probably not been identified by EPA and other Government agencies, pointing out the need for a better means of acquiring information concerning the uses of toxic chemicals. Finally, the problem of unreacted monomer in PVC products has opened a broad vista of possible new concerns associated with contaminants in polymers in general.

REFERENCES

The material presented in this Section is based largely on original investigations and analyses conducted within the Agency. Most of these activities are elaborated in the Appendices which also cite many of the relevant scientific publications. However, the Appendices do not cover the entire range of the activities of the Task Force, and a few additional references concerning this Section are set forth below.

1. Estimates based inter alia on informal communications with the Department of Transportation and Manufacturing Chemists Association.

2. A small sampling of the available literature on the effects of PVC inhalation follows:

   Cylwik, B., "Histological and Histochemical Changes of the Liver in Experimental Polyvinyl Chloride Pneumonoconiosis," Rocz. Akad. Med. im. J. Marchlewskiego w Bialymstoku 17: 93-111, 1972. (Translation).

   Popow, J., "Influence of Polyvinyl Chloride (PVC) Dust on the Respiratory System in the Rat," Roczniki Akademii Med. im. Juliana Marchlewskiego w Bialymstoku 24: 5-48, 1969 (Translation).

   Szende B. et al, "Pneumoconiosis Developing after Inhalation of Polyvinyl Chloride," Orv. Hetil. 112: 85-6, January 10, 1971.

   Wooley, W. D., "Toxic Products from Plastics Materials in Fires." Plastics & Polymers 41 (No. 156), 280-286, December 1973.

3. Bernhard, M. and A. Zattera, "The Importance of Avoiding Chemical Contamination for Successful Cultivation of Marine Organisms," 1970. Helgolander Wiss. Meeresunters. 20:655-675. Also, informal communications with Bureau of Sport Fish and Wildlife, Department of Interior.

4. Jernelov, A., R. Rosenberg, and S. Jensen, "Biological Effects and Physical Properties in the Marine Environment of Aliphatic Chlorinated By-products from Vinyl Chloride Production," Water Research 6: 1181-1191.

5. See also:

   Frey, H. E., "Polyvinyl Chloride Resins," Chemical Economics Handbook, Stanford Research Institute, September 1973.

   Modern Plastics, May 1974, pp. 44-46.

INTERESTS AND ACTIVITIES OF GOVERNMENT AGENCIES AND INDUSTRY

EPA Regulatory Authorities

Pesticide Registration

On April 26, 1974, the Agency determined that the potential risk associated with continued use of VC in pesticidal sprays was unjustified, given the acceptable substitutes which were available. Therefore, on that date Notice was given of the emergency suspension, and intent to cancel the registrations, of all spray products containing VC for use in the home, food handling establishments, hospitals, or other enclosed areas. At the same time EPA requested that all existent stocks of such products be recalled by the manufacturers (39 FR 14753).* In addition, EPA will no longer register pesticides containing VC for indoor or outdoor uses, and all existing registrations have now been withdrawn or amended to substitute another propellant.

At present a few implementing details of this determination remain. Perhaps the most important action is to insure prompt removal of the cans containing VC from commercial channels and environmentally sound disposal of these products, i.e. proper land disposal. EPA Regional Offices have been actively pursuing this problem and should complete their efforts within several months.

Air Pollution

The results of the initial monitoring efforts clearly document the fact that neighborhoods adjacent to PVC resin manufacturing complexes are being exposed to some level of VC air emissions. At present there is no scientific evidence to indicate that these emissions pose an imminent hazard to people living near these plants. However, because of the severe health effects associated with occupational exposure to VC and the lack of data regarding the levels at which effects begin to occur, prudence dictates that steps should be promptly taken to reduce the emissions to the lowest practical level. Indeed, EPA, together with state and local air pollution authorities, has an immediate responsibility to insure that these levels are reduced.

The key questions revolve around (a) the levels of VC concentration that should be achieved outside the plant area, and (b) the regulatory approach that is most appropriate, e.g. ambient air standard, performance standard for new sources, or emission standard for hazardous air pollutants. Several factors bear on such determinations in addition to the uncertainties inherent in the health risks involved, namely: the effect of regulations which are to be promulgated in early October by the Department of Labor on VC levels in the workplace; the technical feasibility, costs, and timing of control technologies and other approaches to reducing emissions; and compliance schedules for the industry as a whole and for individual plants.

**The FR citations identify the Volume and Page of the appropriate Federal Register announcement.*

Depending on the air emission level and/or the level of technology to be achieved, the costs of compliance could have several effects: The price of PVC could increase, thus opening the way for substitutes in some products. Some of the older plants might find it more attractive economically to close or to replace the old technology with new. New plants presumably would emphasize even more than at present larger reactors with fewer requirements for entry and other innovations to reducing leakage rates.

On May 31, EPA requested the manufacturers of VC and PVC resin to provide detailed technical and economic information concerning steps that have been and could be taken to reduce VC emissions. The information was sought under Section 114 of the Clean Air Act.

The Task Force believes that a standard based upon achieving a specific air quality level will be very difficult to develop using currently available data. On the other hand a performance or emission standard, which is designed to drive down the emissions as low as practical, and in the longer run drive technology toward more environmentally acceptable approaches, would seem consistent with the desirability of reducing risks from carcinogens to the minimum possible level.

Present preliminary estimates are that emissions can be reduced by 75 percent in PVC plants and 90 percent in VC plants. Such reductions can be achieved by employing best available control technology which includes a variety of control measures that could be in place from within several months to two years after promulgation of regulations. Each plant would likely use different combinations of such measures to achieve the necessary reductions. Currently available information indicates that such reductions should result in ambient air levels which present no established risk to health or welfare. The cumulative costs of the control technology are estimated to increase the cost of PVC resin by about four percent.

An expanded monitoring program to gain additional data at selected plants to assist in setting an air standard is needed. Twenty-four hour integrated samples taken during a period of several days or longer at a number of sites around representative complexes are desirable. To the extent possible in-plant activities should be correlated with external readings. Since no previous monitoring was done at fabrication or copolymer plants, they should also be included.

Water Pollution

In view of its low solubility and volatility from water, VC is not currently a candidate for the hazardous substances list under Section 311 (spills) of the Federal Water Pollution Control Act. Similarly, VC in water has not been shown to present a threat to aquatic life, and therefore there is no basis at present to develop water quality criteria under Section 304(a) or to consider designating VC as a toxic effluent. At the same time, in view of the monitoring data indicating discharges up to a level of 20 ppm of VC from at least two PVC resin plants, further investigations of its effect, if any, on the aquatic environment seem in order.

The Effluent Guidelines for the Plastics Industry promulgated by EPA cover only PVC polymerization activities and not compounding or fabrication activities. However, some of the potentially most troublesome water effluent problems relate to the toxic materials used in the compounding process.

Also, as previously mentioned, the possibility of toxic additives in PVC products leaching into the aquatic environment is of concern. Further clarification of these problems is needed prior to considering regulatory action.

Solid Waste Disposal

Problems associated with disposal of PVC products through incineration and burial are discussed in detail in Appendix VIII. The emission of HCl is the only clearly identified incineration problem at present although there are many uncertainties concerning the fate of PVC additives during the incineration process. There are well accepted environmentally sound landfill techniques that should adequately contain PVC products. At the same time there are sufficient uncertainties -- particularly with regard to additives -- in both of these areas to warrant more detailed investigations in the months ahead. There is no evidence at present that PVC will revert to VC during incineration or as the result of biological or chemical activity during environmental exposure.

Disposal of the solid and semi-solid wastes from VC/PVC plants poses many problems encountered with hazardous wastes in general. One particular concern is the disposal of tars -- a concern that was heightened last year when a large number of cattle were contaminated due to poor handling and disposal practices involving hexachlorobenzene wastes.[1] Another problem is the possible exposure of personnel at landfills to sludges containing up to 3000 ppm of entrapped VC which might be escaping. Given the widespread concern at the local level over VC/PVC activities, EPA guidance on handling such hazardous wastes would be welcomed. In the longer term the steps called for in the proposed Hazardous Waste Management Act might be particularly appropriate.

Ocean Dumping

The October 1973 criteria for evaluating ocean dumping permit applications prohibit dumping of organohalogen compounds, except for narrowly defined trace amounts, and compounds which may combine with other substances to form organohalogens in the marine environment. These criteria cover the principal earlier concerns over ocean disposal of the by-products of VC/PVC manufacturing activities which were triggered by reports of fish kills in the North Sea from disposal of tars and of the buildup of organohalogen compounds near Puerto Rico. Unfortunately, earlier ocean disposal practices for tars in the Caribbean also have had adverse effects on marine life.[2]

Drinking Water

Currently there are no data on VC concentrations in community water supplies, primarily because no one has ever looked for VC. Conceivably, it could be present in drinking water. Possible sources are PVC polymerization plants just upstream from water intakes and from PVC products used in the distribution system such as PVC pipe and storage tank liners.

Monitoring a few selected water supplies should provide a basis for assessing VC concentrations that may be found in water supplies from both industrial pollution and product contamination. Adequate sampling and analytical procedures will of course be required to insure the soundness of the results. The results should be used to determine the need for additional sampling and possible research requirements.

Proposed Toxic Substances Control Act

An important authority which is currently missing is the Toxic Substances Control Act. The requirements for reporting of industrial production data evisaged in the Act would enhance knowledge of the types and extent of different uses of VC. The testing provision would be the basis to obtain much needed data -- and particularly data on toxicity and persistence -- for assessing the risks associated with low concentration levels of VC, including those levels that are likely to persist beyond the workplace. The proposed regulatory provisions would provide a mechanism for addressing those products using VC not now subject to regulation under other laws. Also, if considered appropriate, steps might be taken to limit the amount of unreacted VC in certain PVC products which could eventually migrate out of these products to pose an unnecessary risk.

Supporting Research Activities

As a component of the national effort to clarify the health risks associated with VC, the Agency plans to support a toxicological effort at the University of Cincinnati. These studies are designed to determine the effects of VC on the developing fetus, to determine changes of VC toxicity caused by nutritional imbalance and interactions with other common chemicals, and to develop cell culture techniques for rapid screening of the oncogenic potential of VC and related compounds. Also, epidemiological studies of neighborhoods near PVC activities are scheduled to complement related efforts of other Government agencies.

Regulatory Interests of Other Agencies

Aerosol Sprays

Early this year evidence indicated that some supplies of hair sprays containing VC were still on the market, and the Food and Drug Administration (FDA) requested that all known manufacturers recall these supplies. In early April, three manufacturers initiated recalls for hair spray and other drug and cosmetic aerosol products. On April 22, FDA published a notice of proposed amendments to the Federal Food, Drug and Cosmetic Act concerning the use of VC as an ingredient, including propellant, of (a) aerosol drug products, and (b) cosmetic aerosol products (39 FR 14215). At the same time, but in a separate notice, FDA requested a list of all marketed drug products containing VC as an ingredient or packaged in containers of or lined with PVC (39 FR 14238). On August 26, FDA published regulations which (a) banned the use of VC in cosmetic aerosol products, and (b) required a new drug application as a condition for marketing drug aerosol products when VC is used as an ingredient (39 FR 30830).

The Consumer Product Safety Commission has undertaken two steps. An information gathering process was initiated in May to determine the specific aerosol products containing VC which are being or have been used (39 FR 16511). The Commission has identified more than 25 aerosol products containing VC and is currently continuing its analyses. On August 21, the Commission promulgated a regulation, effective from October 7, banning as hazardous substances "self-pressurized products intended or suitable for household use that contain VCM as an ingredient or in the propellant" (39 FR 30114).

Worker Protection

The Occupational Safety and Health Administration (OSHA) of the Department of Labor, responsible for worker safety, determined that VC levels exceeding 50 ppm in the workplace present a hazard and on April 5 set an emergency temporary standard at that level (39 FR 12342). To initiate the process for setting a permanent standard, on May 10, OSHA proposed lowering the standard to the level of detection, defined at 1 ppm plus or minus 0.5 ppm (39 FR 16896). Public hearings to gather information for a permanent standard and to assist OSHA in making a final judgement as to the adverse effects of VC and the appropriate levels of exposure to workers were held in June and July. The final standard is to be set by October 5, 1974.

Much of the information being developed by OSHA is of direct relevance to EPA concerns, and particularly concerns over an air standard. Even more importantly the regulatory action taken by OSHA within the next several months can have a profound effect on the need for and character of action by EPA. If OSHA uses health data as the basis for its standard, it is important that the interpretation of the data not be inconsistent among agencies. Also, OSHA should be encouraged to recommend control techniques which will not simply vent VC to the environment external to the workplace.

Unreacted VC Monomer in PVC Products

Early last year reports were received of possible migration of VC to distilled spirits and wines packaged in PVC bottles under an experimental program authorized by the Department of Treasury. Subsequent investigations by FDA confirmed that residual VC had migrated from the PVC into various distilled spirits and wines. Since there were no available toxicological studies supporting a safe level of VC in food, FDA published a proposal which in essence would preclude the use of PVC resin in contact with alcoholic food (39 FR 12931). At the same time, the Department of Treasury withdrew the approval of the experimental use of PVC bottles to contain distilled spirits. Since there was no indication, at that time, that VC migration occurred from PVC in contact with non-alcoholic foods, FDA proposed a regulation which identified criteria for safe use under the prior sanction (38 FR 12931). Recently, however, FDA has received data confirming VC migration from PVC packaging into a variety of foods and currently is considering limitations on the use of PVC in food packaging.

The Consumer Product Safety Commission has not taken a position on unreacted monomer in consumer products.

Transportation Handling

The problems associated with VC have heightened the concern of the Department of Transportation (DOT) as to whether any changes in its regulations are warranted since some commercially important chemicals have been identified as carcinogens. A major question is whether a single exposure to these chemicals can cause cancer. At present, DOT is considering the desirability of changes in labeling requirements and/or packaging requirements for carcinogens but has not yet reached a conclusion.

In July 1974, DOT published (a) proposals to amend the bulk dangerous cargoes regulations for the carriage of VC (39 FR 26752), and (b) a requirement for protective head shields on uninsulated tank cars carrying liquefied flammable compressed gases such as VC (39 FR 27572). In a related action in August, DOT proposed amending the requirements for handling freight cars carrying hazardous materials to include those placards as "Dangerous" (39 FR 29197).

Related Research Activities

The Center for Disease Control (CDC) and the National Institute of Occupational Safety and Health (NIOSH) are maintaining a nationwide surveillance network of all cases of angiosarcoma of the liver which have been reported since 1965. For each case identified, the health history of the individual will be reviewed and analyzed to determine if there was any connection with VC or PVC plants. CDC/NIOSH are also conducting studies to determine if other cancers and other mortality causes are associated with exposure to VC. In these studies, pathological information will be obtained from hospitals. A CDC survey among meat wrappers in Houston, Texas, should determine if chemicals produced upon combustion of PVC film may be implicated in "meat wrappers asthma."

CDC/NIOSH are conducting a comprehensive epidemiological survey of workers from 11 polymerization and fabricating plants. Employee medical records are being subjected to intense screening to determine the scope and magnitude of VC effects. Hospital data obtained in this study are being sent to the angiosarcoma network. NIOSH is also planning studies to determine the levels of exposure at fabricating plants and to determine if cosmeticians exposed to VC from aerosol hair sprays have had any cases of liver angiosarcoma.

The Consumer Product Safety Commission plans to support rodent experiments to determine the effects over a lifetime of single dose and/or intermittent exposures of VC at several dose levels. Reproductive, mutagenic, and teratogenic effects may be considered.

The National Institute of Environmental Health Sciences (NIEHS) has taken an active role in bringing together interested Government agencies, industry, and the academic community to begin to assess the public health implications of a broad range of chemicals used in the plastics industry. An initial meeting in Pinehurst, North Carolina, from July 29 to 31 was designed to set the stage for a continuing effort to sort out research priorities within Government and industry, as well as to highlight the types of considerations that should surround regulatory actions in this field.

The National Cancer Institute is providing pathology services (human and experimental animal) and in collaboration with the Armed Forces Institute of Pathology is establishing a case registry for VC associated diseases.

The National Bureau of Standards (NBS) is conducting research on the development of calibration methods for EPA. This includes preparation of standard VC air mixtures in the ppm range and of charcoal sampling tubes with known VC loadings. NBS is also doing research to develop more sensitive techniques for the detection of VC in the atmosphere.

Role of Industry

During the past several months industry has cooperated extensively with the Task Force and with individual Agency offices in the assessment of problems associated with VC/PVC activities. Summarized below are some of the areas of greatest concern involving major industrial commitments.

Reducing Disharges of VC and Other Toxic Chemicals

There is no doubt that industry has taken and can continue to take a variety of immediate steps at relatively little cost to reduce the VC losses at VC and PVC polymerization facilities. During the past few months many plants have already started to tighten maintenance and operating procedures; other plants are installing improved pumps, seals, and disconnect devices; while still other plants are introducing more significant process changes. One company is reportedly spending $3 million to tighten the processes at a single PVC facility; another company reports that it has 100 engineers working to introduce modifications that will dramatically cut VC losses at several plants.

In the longer run significantly different approaches to the polymerization process may be in order. Clearly, the opening of the reactor kettles is a major source of discharges, and a continuous rather than batch process should be considered as an example of a design change to reduce VC losses. In the batch process the trend toward larger kettles will probably accelerate. At the same time some companies may elect to mark time with regard to major modifications or significant new departures until the initial OSHA and EPA regulatory approaches become clearer.

A number of other toxic chemicals are also associated with VC/PVC activities. As previously mentioned the disposal of tars and the discharges of toxic metals at PVC compounding plants are of particular concern. A number of the troublesome chemicals may be better known to industry than Government, and industry should not delay in taking corrective actions even though these chemicals have not yet been designated for control by the Government.

Medical Surveillance

Clearly, VC concerns have triggered extensive medical surveillance programs of VC and PVC workers throughout the industry. These programs should become routine to cover a far broader swath of chemicals at VC/PVC and other chemical complexes. Published analyses of the results of such programs would be very valuable to EPA and other agencies.

In addition, industry has a responsibility to support medical surveillance programs for residents in neighborhoods adjacent to VC/PVC complexes and other types of plants releasing chemicals into these neighborhoods. The character of such surveillance will obviously depend on the type of chemicals involved and the arrangements that can be worked out by industry with local health authorities.

Fenceline Monitoring for Chemical Discharges

Traditionally, the chemical industry has conducted very little fenceline monitoring not required by Federal, state, or local agencies to determine the chemical discharges leaving plant property. During the past several months, however, monitoring for VC has become a concern, and hopefully this concern will rapidly spread to other chemicals. Clearly, a plant manager should know the chemical mix of the air emissions drifting over the plant fence into nearby neighborhoods. Similarly, he should be fully aware of the chemical cross section of his liquid and solid waste streams. Thus, a far more intensive physical monitoring effort on the part of industry is needed -- monitoring not only for gross pollution (e.g., biological oxygen demand, chemical oxygen demand, total suspended solids) but for individual chemicals as well, including VC and related chlorinated hydrocarbons.

Toxicological Testing of VC

Until the recent revelations concerning the relationship of VC to angiosarcoma of the liver, the efforts of U.S. industry to clarify the chronic toxicity of VC were nearly negligible, despite the commercial importance of VC. The industrial studies of the early sixties and the recent Manufacturing Chemists Association (MCA) toxicological study on behalf of a number of companies have not been adequate, in terms of direction, scope, or quality. Even the additional toxicological studies which have been proposed by MCA calling for animal exposures down to 1 ppm of VC may not be sufficient if the objective is to understand in some detail the biological effects from the types of dose levels likely to be found in the workplace and beyond. This proposed effort, while important, is but a small step toward a very complicated problem. Also, a number of experts have expressed concern over the design and statistical significance of the studies as currently planned.

Testing for Persistence and Environmental Fate and Effects of VC and PVC

A related area is industry's responsibility to clarify the environmental fate and effects of the chemicals it manufactures, and in this case the behavior of VC in water and air (including degradation products) and the fate and effects of products containing PVC in soil and water. This is a new area which has not attracted sufficient attention from industry but which is of crucial importance in assessing environmental impacts of chemical activities. Governmental leadership will probably be essential in helping to point the way as to the types of tests and analyses that are the most appropriate.

Testing for Levels of Unreacted Monomer in PVC Resins and PVC Products

In view of the likelihood that FDA will limit the levels of unreacted VC allowed in PVC food packaging, industry has recently accelerated efforts to analyze the levels of VC that are present in PVC resin used for food packaging and in the packaging itself. This relatively inexpensive procedure should be extended to other types of products as well. It is particularly important that the manufacturers of resin, who in general are well equipped to carry out the necessary sampling and analysis, advise their customers (i.e., the fabricators) of the quality of the resin in terms of unreacted monomer in addition to the usual quality criteria. The fabricators in turn have a responsibility to be aware of the levels of unreacted monomer that persist in the products that eventually reach the marketplace.

REFERENCES

The technical information presented in this Section is based principally on informal communications with the concerned EPA offices, other Federal agencies, and industrial representatives. Two relevant publications not cited in the Appendices are identified below:

1. Environmental Contamination from Hexachlorobenzene, Office of Toxic Substances, Environmental Protection Agency, July 20, 1973.
2. Aubert, M., "Effect on the Marine Environment of the Combustion at Sea of Some Industrial Waste", Center of Biological Studies and Research and of Oceanographic Medicine (C.E.R.B.O.M.), Nice, France, January 1974.

RECOMMENDATIONS

This Section sets forth the recommendations of the Task Force concerning steps that should be taken by the Agency and steps that the Agency should encourage industry to take in the near term to (a) help clarify and reduce the risks associated with VC/PVC activities, and (b) take full advantage of our experiences with these chemicals in addressing other chemicals.

Regulatory and Directly Supportive Actions by EPA

Recommendation #1:

An air standard for VC should be established as soon as practical under the Clean Air Act for VC and PVC polymerization plants and, if warranted by further investigations, for PVC fabrication plants. The Agency should determine the ambient levels that are likely to be achieved and, to the extent possible, the health risks associated with such levels

Recommendation #2:

Additional ambient air monitoring should be carried out to support regulatory action under the Clean Air Act. These efforts should include sampling at a number of carefully selected sites around a few VC, PVC polymerization, and PVC fabrication plants. The monitoring measurements made at these facilities should be correlated with specific in-plant activities such as reactor venting.

Recommendation #3:

More detailed material balance studies should be conducted, in cooperation with industry, at a few VC and PVC polymerization plants. Specific VC leakage points should be more clearly identified, and attempts should be made to correlate the magnitude and timing of the estimated losses with the levels of VC detected in a monitoring program.

Recommendation #4:

A program should be initiated to determine whether and to what extent background levels of VC are present in the ambient air -- indoors, and outdoors--due to the presence of PVC products.

Recommendation #5:

A limited VC monitoring program should be undertaken of drinking water supplies which might be contaminated from VC discharges from nearby PVC plants. Prior to undertaking the program sampling and analysis procedures should be carefully reviewed and refined as necessary.

Recommendation #6:

A study should be conducted to determine the amount of VC migrating out of PVC products used in water distribution systems--such as PVC pipe or storage tank liners. Prior to undertaking the program sampling and analysis procedures should be carefully reviewed and refined as necessary.

Recommendation #7:

To insure comparability of results between laboratories EPA should further develop its interim method into a standardized method for monitoring levels of VC. Concurrently, the Agency should also investigate the feasibility of developing continuous air monitoring devices.

Recommendation #8:

Monitoring and bench-scale studies should be conducted around industrial storage and disposal sites and municipal disposal sites to determine the types and quantities of toxic substances leached or discharged out of (a) semi-solid and solid wastes generated by VC/PVC facilities, or (b) PVC products discarded by consumers. If these studies indicate that there could be a health or environmental hazard, guidelines should be developed to control the storage and disposal of these wastes.

Recommendation #9:

The responsible Office should continue to support currently planned VC toxicological studies.

Recommendation #10:

The responsible Office should continue to support currently planned VC epidemiological studies.

Recommendation #11:

More intensive studies should be conducted on the behavior of VC in the atmosphere and in the aquatic environment, and particularly on its degradation products and related chemical reactions. These studies should be supported by both industry and Government.

Recommendation #12:

The industries covered by the Effluent Guidelines for the Plastics Industry promulgated under the Federal Water Pollution Control Act should be expanded beyond the production of resins to include the compounding and directly associated activities that result in discharges into the water of toxic metals and other chemicals of particular concern.

Recommendation #13:

Enforcement efforts, including spot checks of manufacturers, distributors, and retail outlets, should continue to be pursued vigorously to insure that pesticidal sprays containing VC as a propellant are removed from the channels of trade as rapidly as possible.

Recommendation #14:

Regional, State, and local authorities should be kept fully informed of Agency efforts under the Clean Air Act and other authorities. As the Federal approach becomes clearer, they should be encouraged to undertake supportive actions as appropriate.

Recommendation #15:

The Agency should build on the experience gained in responding to the problems associated with VC in strengthening its organizational, manpower, and contractor resources to anticipate and respond to similar situations involving other chemicals. Readily available information on the handling of VC should be made available to the Regional Offices to assist them in responding to rail or barge accidents resulting in the release of VC.

Recommendation #16:

Laboratory procedures for safe handling of VC should be developed and distributed to all EPA laboratories. Consideration should be promptly given to how such procedures can most effectively be developed for a number of carcinogens that are likely to be of concern to the Agency.

Recommendation #17:

Should the Toxic Substances Control Act be enacted, prompt consideration should be given to the need for and feasibility of (a) requirements for industrial testing of the toxicity of VC at low ambient levels and the persistence of VC in different media, and (b) limitations on the levels of unreacted VC monomer in selected PVC products.

Recommendation #18:

The Agency should continue its leadership role in bringing together the interested Federal agencies to exchange views on regulatory actions, supporting activities, and research projects directed to problems associated with VC and PVC. In addition, an appropriate interagency mechanism should be developed to address a broader spectrum of potential problems associated with the plastics industry.

Recommendation #19:

EPA should exercise leadership in stimulating Governmental and industrial efforts to analyze in depth the other high volume chemicals (e.g., top 50 in terms of pounds of production) to identify those which deserve additional testing or controls to clarify or reduce potential environmental problems.

Steps by Industry

Recommendation #20:

All possible steps to tighten up operating and maintenance procedures to reduce VC and PVC losses at VC, PVC polymerization, and PVC fabrication facilities should be taken promptly without waiting for further Governmental regulatory actions. R&D efforts should be expanded to develop new approaches to reduce losses during polymerization, such as continuous flow systems.

Recommendation #21:

Each VC and PVC resin facility, and indeed chemical complexes in general, should have up-to-date information on the character and extent of the chemical pollutants that are leaving the plant property as air, water, or solid waste discharges or degradation products. A systematic monitoring program operated by industry at the fence line and beyond will in many cases be essential to ascertain the nature of the pollutants reaching nearby neighborhoods.

Recommendation #22:

The levels of unreacted VC monomer in all grades of PVC resin should be routinely ascertained and purchasers of the resin should be advised accordingly. Also, analyses of the rates of release of VC monomer from PVC products should be expanded.

Recommendation #23:

The Manufacturing Chemists Association should, in consultation with interested Government agencies, carefully reevaluate its planned VC toxicological experiments at low doses to insure that the design is (a) statistically reliable, and (b) relevant to the ambient air concerns of EPA.

PRELIMINARY ASSESSMENT OF THE ENVIRONMENTAL PROBLEMS ASSOCIATED WITH VINYL CHLORIDE AND POLYVINYL CHLORIDE

(Appendices)

Report on the Activities and Findings of the
Vinyl Chloride Task Force

Environmental Protection Agency
Washington, DC
September 1974
30<

TABLE OF CONTENTS

APPENDICES

I. Selected Economic Considerations 1

- Production Levels 1
- Competitive Substitution 1
- International Aspects 4
- Control Technology 4

II. Producers of Vinyl Chloride and Polyvinyl Chloride 6

- VC Producers 6
- PVC Producers 6
- PVC Copolymer Producers 8

III. The Materials Balance at Vinyl Chloride and Polyvinyl Chloride Facilities 10

- Vinyl Chloride Production Facilities 10
- Polyvinyl Chloride Polymerization Facilities 11

IV. Interim Method for Sampling and Analysis of Vinyl Chloride in Waste Water Effluents and Air Emissions 17

- Scope and Application 17
- Summary of Analytical Procedures 17
- Interferences 17
- Apparatus and Materials 18
- Reagents, Solvents, and Standards 19
- Sampling 20
- Calibration 23
- Procedure 25
- Quality Control 25

V. Summary of Regional Activities 26

- Region I: Leominster, Massachusetts 26
- Region II: Flemington, New Jersey 27
- Region III: Delaware City, Delaware 27
  S. Charleston, W. Virginia
- Region IV: Louisville, Kentucky 28
- Region V: Painesville, Ohio 28
- Region VI: Plaquemine, Louisiana 29
- Region IX: Long Beach, California 29

31<

VI. Persistence of Vinyl Chloride 31

- Behavior of Vinyl Chloride in Air 31
- Behavior of Vinyl Chloride in Water 31
- Behavior of Vinyl Chloride in Closed Rooms

VII. Health Effects of Vinyl Chloride 32

- Occupational Cases of Liver Angiosarcoma 34
- Cases of Hepatic Angiosarcoma, Connecticut, 1935-1973 38
- Observed Deaths/Expected Deaths in VC Workers 40
- Summary of Toxicological and Epidemiological Studies on Vinyl Chloride 44

VIII. Disposal of Products Containing Polyvinyl Chloride 63

- Incineration 63
- Landfilling 64
- Resource Recovery 65

IX. Activities of Task Force 67

32<

SELECTED ECONOMIC CONSIDERATIONS

Production Levels

During 1973, VC production was at the 5.3 billion pound level with PVC and its copolymers at the 4.6 billion pound level. PVC has become a very important polymer as evidenced by the broad dependence of nearly every branch of industrial and commercial activity upon products and components fabricated from this plastic. In Table 1, major PVC products manufactured during 1973 are identified.

The U.S. VC/PVC industry has been operating for more than forty years, and over the past five years has shown an average annual growth rate of 14 percent -- a rate of growth that had been expected to taper off only moderately in the next few years.

The size of this industry can be appreciated by considering that the synthesis of the monomer is conducted in fifteen U.S. plants, and forty-three facilities are engaged in polymerization of PVC (including its use as a copolymer) with almost all of these plants currently operating at or near capacity. At least 7,500 plants are engaged in fabricating products from PVC. About 1,500 workers are employed in monomer synthesis and an additional 5,000 in polymerization operations. Estimates have suggested that up to 350,000 workers may be associated with the fabrication plants.

The wholesale value of the annual output of fabricated products based on PVC is at least several billion dollars.

Competitive Substitution

Should requirements for worker safety or environmental controls drive the price of PVC resin upward, it seems likely that some PVC products would be displaced by products using other plastics or other materials. Other products dependent on PVC might disappear altogether from the marketplace. Probably one-fourth to one-third of current PVC products by value are marginally competitive with other plastic products. At significantly higher prices a lesser number probably would find substitutes in other materials at higher costs. Identified in Table 2 are a few of the substitute materials that might be considered. For some uses, there are no apparent substitutes.

Table 1
MAJOR PVC PRODUCTS

Market Category		Products	1973 1000 metric tons
I.	Apparel	Baby pants	12
		Footwear	66
		Outerwear	31
II.	Building and Construction	Extruded foam moldings	26
		Flooring	211
		Lighting	5
		Panels and siding	39
		Pipe and conduit	525
		Pipe fittings	44
		Rainwater systems, soffits, facias	16
		Swimming pool liners	18
		Weatherstripping	16
		Windows	26
III.	Electrical	Wire and cable	194
IV.	Home	Appliances	20
		Furniture	145
		Garden hose	18
		Housewares	51
		Wall coverings and wood surfacing films	54
V.	Packaging	Blow molded bottles	36
		Closure liners and gaskets	9
		Coatings	9
		Film	59
		Sheet	35
VI.	Recreation	Phonograph records	66
		Sporting goods	25
		Toys	88
VII.	Transportation	Auto mats	18
		Auto tops	15
		Upholstery and seat covers	83
VIII.	Miscellaneous	Agriculture (incl. pipe)	66
		Credit cards	8
		Laminates	23
		Medical tubing	23
		Novelties	7
		Stationery supplies	18
		Tools and hardware	8
		Other	45
		Total 34<	2158

Table 2
SUBSTITUTE MATERIALS FOR PVC PRODUCTS

PVC PRODUCT	SUBSTITUTES	SAME PRICE RANGE	HIGHER PRICE
Pipe & Tubing	Polyethylene	X	
	Polypropylene	X	
	Metals		X
	ABS resins		X
Flooring	Asphalt	X	
	Wood		X
	ABS resins		X
Electrical Insulation	Polyethylene	X	
	Polypropylene	X	
	EPDM rubbers		X
	SBR rubbers		X
	TFE plastics		X
Records	ABS resins		X
	Acrylics		X
Film & Sheet Products	Polyvinylidene chloride		X
	Polyethylene	X	
	Polypropylene	X	
	Cellulosics		X
Coatings	Acrylics		X
	Polyurethanes		X
	Cellulosics		X
Household Goods	Styrene	X	
	Polyethylene	X	
	Polypropylene	X	
	Wood		X
	Metals		X
	Acrylics		X
Packaging	Polyethylene	X	
	Polypropylene	X	
	Polyvinylidene chloride		X
	Cellulosics		X
	Acrylics		X
	Polyurethanes		X
	Glass		X

International Aspects

U. S. based manufacturers currently produce about one-third of the western world's supply of resins, with the U. S. market also consuming about one-third of the total. In 1973, 3.7 percent of PVC and 7.8 percent of VC manufactured in the United States were exported. Prior to the recent U. S. concern over worker and environmental controls at VC and PVC facilities, there was no reason to anticipate a major change in the U. S. share of production or market during the next few years. Recent increases in demand for PVC resins -- and concurrently for VC -- at attractive prices have been of worldwide dimensions with expansion plans for PVC manufacturing being considered by a number of companies at home and abroad.

There is presently an import duty on PVC resin from countries with status as Most Favored Nations of 1 1/4 cents per pound plus six percent ad valorem and from other nations of four cents per pound plus 30 percent ad valorem. Given the current U. S. market price of 18 to 24 cents per pound for the general purpose uncompounded resin, there has been little incentive to import PVC resin. Also, there currently is little export incentive because of short U. S. supply and unattractive foreign prices. However, higher prices as a result of more stringent worker or environmental controls in PVC resin plants in the United States than abroad might well stimulate significantly increased imports.

Control Technology

While there appear to be a number of general approaches for reducing the discharge of VC into the environment at VC and PVC resin plants and the discharge of PVC at resin plants, in many respects the approaches must be tailored to the individual plants. All VC plants and some PVC resin plants are outdoors while other PVC plants are at least partially enclosed. A variety of production processes are used, and different kinds of technology are employed. However, there are some common measures that would reduce VC emissions.

FOR VC PLANTS:

1. Reducing the escape into the atmosphere of VC when venting the tank car gauge tube, disconnecting the feeding line, and closing the valves during rail tank car loading. Mechanical disconnect devices and double block and bleed piping are available to ease this problem.

2. Improving the quality of pumps to reduce the possibility of leakage due to failure of seals. Pumps are available today which could minimize this problem.

3. Venting unintentional leaks and spills into a system which is flared and, preferably, scrubbed.

FOR PVC RESIN PLANTS:

1. Collection and destruction of purge gases from the reaction kettles prior to opening for cleaning, sampling, or recharging.

2. Centralized collection and filtering of VC vapor discharges from dryers and centrifuges.

With regard to PVC particulate in air and water discharges, improved housekeeping and relatively simple ventilation filtering systems are usually technically feasible and effective.

Laboratory data have shown that VC can be adsorbed on activated carbon. Concentrated VC vapor streams have produced a recovery working capacity on carbon equivalent to about ten percent of the carbon weight. Ambient air contaminated with low levels of VC produces significantly lower adsorbent working capacities. Control of dilute VC is therefore possible but may not be practical using activated carbon. Carbon regeneration using steam or pressure swing appears possible, with recovery of desorbed VC for recycle.

Clearly, these approaches will not eliminate losses but should materially reduce them. In the longer run, the development of continuous flow processes, the use of larger kettles, better housekeeping, and/or reductions in the number of feed lines might result in more dramatic reductions of VC leakage.

REFERENCES

1. Modern Plastics, Jan 1974, p. 43

2. The 1972 Census of Manufacturers shows 7,574 plants manufacturing miscellaneous plastics products (SIC 3079), a substantial number of which use PVC. SIC 3079 probably covers most, but not all, PVC fabricators.

3. Discussions with representatives of the Department of Commerce, Manufacturing Chemists Association, and Society of Plastics Industry.

APPENDIX II

PRODUCERS OF VINYL CHLORIDE AND POLYVINYL CHLORIDE

The major producers of VC, PVC, and PVC copolymers are listed in this section with the plant location and available capacity data.

VC Producers

	Location	Annual Capacity (Millions of Pounds)
Allied Chemical Corporation	Baton Rouge, La.	300
American Chemical Corporation	Long Beach, Calif.	175
Continental Oil Company	Westlake, La.	650
Dow Chemical, U. S. A.	Freeport, Tex.	200
	Oyster Creek, Tex.	700
	Plaquemine, La.	390
Ethyl Corporation	Baton Rouge, La.	300
	Pasadena, Tex.	150
B. F. Goodrich Chemical Company	Calvert City, Ky.	1000
Monochem, Inc.	Geismar, La.	300
PPG Industries, Inc.	Lake Charles, La.	400
	Guayanilla, P. R.	500
Shell Chemical Company	Deer Park, Tex.	840
	Norco, Tex.	700
Tenneco, Inc.	Houston, Tex.	225

PVC Producers

Air Products and Chemicals, Inc.	Calvert City, Ky.	150
	Pensacola, Fla.	50
American Chemical Corporation	Long Beach, Calif.	150
Borden, Inc.	Illiopolis, Ill.	140
	Leominster, Mass.	180
Continental Oil Company	Aberdeen, Miss.	285
	Oklahoma City, Okla.	240

38<

Company	Locations	Annual Capacity (Millions of Pounds)
Diamond Shamrock Chemical Company	Deer Park, Tex.	270
	Delaware City, Del.	100
Ethyl Corporation	Baton Rouge, La.	180
The Firestone Tire & Rubber Company	Perryville, Md.	230
	Pottstown, Pa.	270
The General Tire & Rubber Company	Ashtabula, Ohio	125
	Pleasants County, W. Va.	50
B. F. Goodrich Chemical Company	Avon Lake, Ohio	140
	Henry, Ill.	140
	Long Beach, Calif.	140
	Louisville, Ky.	340
	Pedricktown, N. J.	170
The Goodyear Tire & Rubber Company	Niagara Falls, N. Y.	100
	Plaquemine, La.	100
Great American Chemical Corporation	Fitchburg, Mass.	40
Hooker Chemical Corporation	Burlington, N. J.	180
	Hicksville, N. Y.	15
Keysor-Century Corporation	Saugus, Calif.	35
	Delaware City, Del.	35
Monsanto Company	Springfield, Mass.	70
National Starch & Chemical Corporation	Meredosia, Ill.	10
Olin Corporation	Assonet, Mass.	150
The Pantasote Co. of New York, Inc.	Passiac, N. J.	60
	Point Pleasant, W. Va.	90
Robintech, Inc.	Painesville, Ohio	250
Stauffer Chemical Company	Delaware City, Del.	175
Tenneco Chemicals, Inc.	Burlington, N. J.	165
	Flemington, N. J.	70
Union Carbide Corporation	South Charleston, W. Va.	160
	Texas City, Tex.	240
Uniroyal, Inc.	Painesville, Ohio	140

39<

PVC Copolymer Producers

Company	Locations
A. Polyvinyl Chloride-Propylene Copolymer Resins	
Air Products and Chemicals, Inc.	Calvert City, Ky.
B. Polyvinyl Chloride-Vinyl Acetate Copolymer Resins	
Air Products and Chemicals, Inc.	Calvert City, Ky.
American Chemical Corporation	Long Beach, Calif.
Atlantic Tubing & Rubber Company	Cranston, R.I.
Borden, Inc.	Bainbridge, N.Y. Compton, Calif. Demopolis, Ala. Illiopolis, Ill. Leominster, Mass.
The Firestone Tire & Rubber Comany	Pottstown, Pa.
B.F. Goodrich Chemical Company	Avon Lake, Ohio Louisville, Ky.
Hooker Chemical Corporation	Hicksville, N.Y.
Keysor-Century Corporation	Saugus, Calif.
National Starch and Chemical Corporation	Meredosia, Ill.
Olin Corporation	Assonet, Mass.
The Pantasote Company of New York, Inc.	Passaic, N.J. Point Pleasant, W.Va.
C. Polyvinyl Chloride-Vinylidene Chloride Copolymer Resins	
BASF Wyandotte Corporation	South Kearny, N.J.
Borden, Inc.	Bainbridge, N.Y. Compton, Calif. Demopolis, Ala. Illiopolis, Ill. Leominster, Mass.

Dow Chemical, U.S.A.	Midland, Mich.
B.F. Goodrich Chemical Company	Louisville, Ky.
W.R. Grace & Company	Owensboro, Ky. South Acton, Mass.
Morton-Norwich Products, Inc.	Ringwood, Ill.
National Starch and Chemical Corporation	Meredosia, Ill.
SCM Corporation	Huron, Ohio
Tenneco, Inc.	Burlington, N.J. Flemington, N.J.
Union Carbide Corporation	Institute and South Charleston, W.Va. Texas City, Texas

REFERENCES

1. 1974 Directory of Chemical Producers, USA, Chemical Information Services, Stanford Research Institute, Menlo Park, California, 1974.
2. Chemical Marketing Reporter, May 20, 1974.

41<

THE MATERIALS BALANCE AT VINYL CHLORIDE AND POLYVINYL CHLORIDE FACILITIES

Vinyl Chloride Production Facilities

Detailed, reliable data for estimating material losses at VC facilities with precision are not readily available. Therefore, only generalized estimates have been attempted.

A simplified block diagram for production of VC from ethylene and chlorine is shown in Figure 1. Some VC complexes utilize oxychlorination units; others produce ethyl chloride from the by-product hydrogen chloride (HCl) and ethylene. However, the production of dichloroethane (EDC) allows for many approaches to recycling of light and heavy materials such that the losses of VC are reduced. Even vent streams of inerts can be scrubbed with EDC for maximum removal of VC before venting. Light ends such as methane are usually flared and VC is converted to water and small amounts of HCl.

VC losses have come primarily from vent streams, the storage and transportation loading systems, and seepages from pumps. If vent streams are not scrubbed or flared, the amount of VC reaching the atmosphere increases considerably. This in turn is influenced by the purity of the ethylene and the chlorine being fed into the units. Usually, these inerts come out in the EDC unit but may be carried on depending upon the producer's philosophy regarding the purity of the EDC to be fed to the cracker. Experience has been that the higher the purity of EDC both with regard to light and heavy material, the greater the efficiency of the cracking.

It is frequently difficult to pinpoint the areas and quantities of VC losses. However, some generalizations can be made for, as an example, a plant producing 500 million pounds per year of VC. (The industry is heading toward plants of this size and larger.) Tank car loading losses may be several hundred pounds per day. Vent stream losses could reach another 100 pounds per day while losses of VC entrapped in the water effluent might be a few pounds per day. In addition to these very small operating losses, there are undoubtedly unintentional losses from leaking pumps, flanges, and containment vessels, with total plant losses probably less than 0.1% or less than 500,000 pounds per year.

From an environmental standpoint, the disposal of the heavy chlorinated hydrocarbons may also present a problem. Some are sold to solvent scrap dealers for salvage. In the past much of the material has been dumped at sea or put into landfills or deep wells. More recently, incineration has been used, which is known to produce HCl emissions.

Polyvinyl Chloride Polymerization Facilities

Reasonably reliable data are available for estimating material losses at PVC facilities. However, generalizations applicable to the entire industry must be surrounded with many caveats. It must be emphasized that there are a number of PVC processes, and each plant has its own idiosyncrasies.

VC losses will fluctuate depending on the care exercised in operating the PVC plant, types of products produced, frequency of product change, method of PVC shipment, and emergency situations. Estimates of losses have varied widely in the industry, indicating the complexity of establishing precise losses for a given facility and overall losses on a nationwide basis.

In general, older PVC plants are smaller than those being built today and are equipped with smaller sized reactors. With small reactors, the number of batches required to produce a given amount of PVC is greater, and thus the number of process steps are increased with a greater potential for loss of both VC and PVC. Further, a small plant has the disadvantage of having to make frequent resin changes to meet customer demands. During these changeovers a certain amount of off-grade resin is produced.

In addition, older plants have the added burden of higher maintenance than new plants, but this tends to stabilize after a few years. The handling of VC and the production of the high quality resins which are demanded by the marketplace require a reasonable maintenance program. Maintenance consists primarily of the care of agitator seals, pump seals, and valves and the removal of polymer which slowly builds up in VC lines -- primarily in the recovery system. Although many older facilities have been in operation for years, they are usually not the same as when first installed. Some of the operators have continually updated the plants for many reasons including labor savings systems, new product requirements, replacement of wornout equipment, addition of new product lines, and safety.

When VC was cheap and there was little concern about its toxicity, the emphasis was almost exclusively on productivity. Often this resulted in high losses of VC to the environment as recovery cycles were reduced. Today, the picture is changing. Not only are the producers trying to reduce the direct VC losses, but they are also trying to minimize PVC losses by scheduling longer production runs between product changes. As an example, the newer large plants are set up with multiple production lines. This allows the dedication of one line to a given product which results in very low resin loss due to product change.

43<

The traditional method of stating yield of VC in PVC plants has been based upon pounds of prime resin in the bag as compared to VC invoiced. This often has led to a misunderstanding about VC losses with the interpretation that a 94% yield means 6% VC loss to the environment. In fact some VC may never actually be received because of the inability to measure the weight of tank cars accurately, some of the losses are in the form of PVC scrap, and some losses escape as PVC particles.

A properly run and maintained suspension plant using technology that is ten years old should be capable of obtaining a 95% or higher yield unless some especially esoteric resin is being produced along with large amounts of scrap or off-grade resin. For the older plants, the losses will probably be significantly higher. Other than overall sloppy operation, the recovery system is the single most important part of the plant governing VC losses. If insufficient time is allowed or vacuum is not applied, then the VC content in the PVC/water slurry will be greater than necessary. As a result, VC losses will occur in the centrifuge effluent water, drier/ product collector vent air, the venting of the reactor, and the slurry tank.

The magnitude of VC and PVC losses in a typical PVC plant is described in Figure 2. These losses are expressed as a range of losses depending on the feed rate, reactor size, reactor cleaning procedures, batch sizes, level of technology, and general housekeeping and operating procedures.

The following comments on manufacturing practices may help put these losses into perspective:

1. VC Feed - This is shipped as virtually 100% VC and does not normally contain an inhibitor.

2. VC Unloading - Considering normal losses in disconnecting the piping, sampling, tank gauging, pump and compressor seals to the tank cars, losses to the atmosphere should not be greater than 100 pounds per car.

3. VC Charging - A 0.05% loss between storage and polymerization should cover losses from flanges and seals throughout all VC handling equipment.

4. Polymerization - The loss from build-up of PVC on the walls of the reactor is split between reactor wash-out and the slurry strainer.

5. Reactor Venting - Before the reactor can be cleaned, residual VC is vented. After recovery and emptying the PVC resin, the reactor is full of a mixture of air, moisture, and VC at ambient conditions.

6. Recovery - Processing schemes will vary, but one of the most widely used is the direct recovery of unreacted VC from the reactor. While the reaction can be carried out further, economically it is essentially complete at 90% conversion or even less depending on the type of resin. At this point the residual VC is recovered by means of compressors which evacuate VC from the reactor. The recovered VC is condensed and distilled before recycling to the reactor.

7. Drying - Unreacted VC is collected in the recovery system but there are losses of polymer in the drier due to coalescence of the resin and periodic clean-out. This is almost entirely scrap.

8. Product Collector - Most plants use bag collectors so that the loss of resin is less than one pound per hour, but there are losses due to product changes which raise the total.

9. Screening - Oversize resin is removed from the final product. This material consists of scrap and off-grade resin. With the current PVC shortage much of this off-grade resin is used as prime resin by special customers.

10. Miscellaneous - In addition to the above losses, others occur as scrap or off-grade polymer and as quality control samples.

    a. Bad Batches - Most plants experience batches which are off specification. These range from "just slightly off" to solid batches, with losses at 2 to 3 batches per month or about 0.4% or 40 pounds per hour average. Salvage value depends upon the degree of "off-grade" and market conditions.

    b. Samples - Probably about 0.05% or 5 pounds per hour and is usually destroyed in testing.

    c. Polymer Build-up - VC slowly polymerizes in the pipe lines, particularly the recovery system, and must be removed periodically. No quantitative value is available for this loss.

    d. Spillage - Some of the product is shipped in bulk and some is bagged. While some spillage occurs in bulk handling, more occurs in bag filling and in bag breakage.

    e. Centrifuge Effluent - Some PVC enters the effluent water.

11. Product Change-Over - As indicated previously there are losses in the drier and collector due to cleaning for changes from one product to another. In addition one must segregate the first product that comes through this system. The amount can vary widely depending upon the number of changes and the sensitivity of the product to contamination from the previous product.

45<

The foregoing analysis, together with estimates provided by industry, suggests that the losses of VC at PVC polymerization facilities currently range from about 3.0 to 6.3% while PVC losses are on the order of 1.3%.

46<

PRODUCTION OF VC FROM ETHYLENE AND CHLORINE
SIMPLIFIED BLOCK DIAGRAM

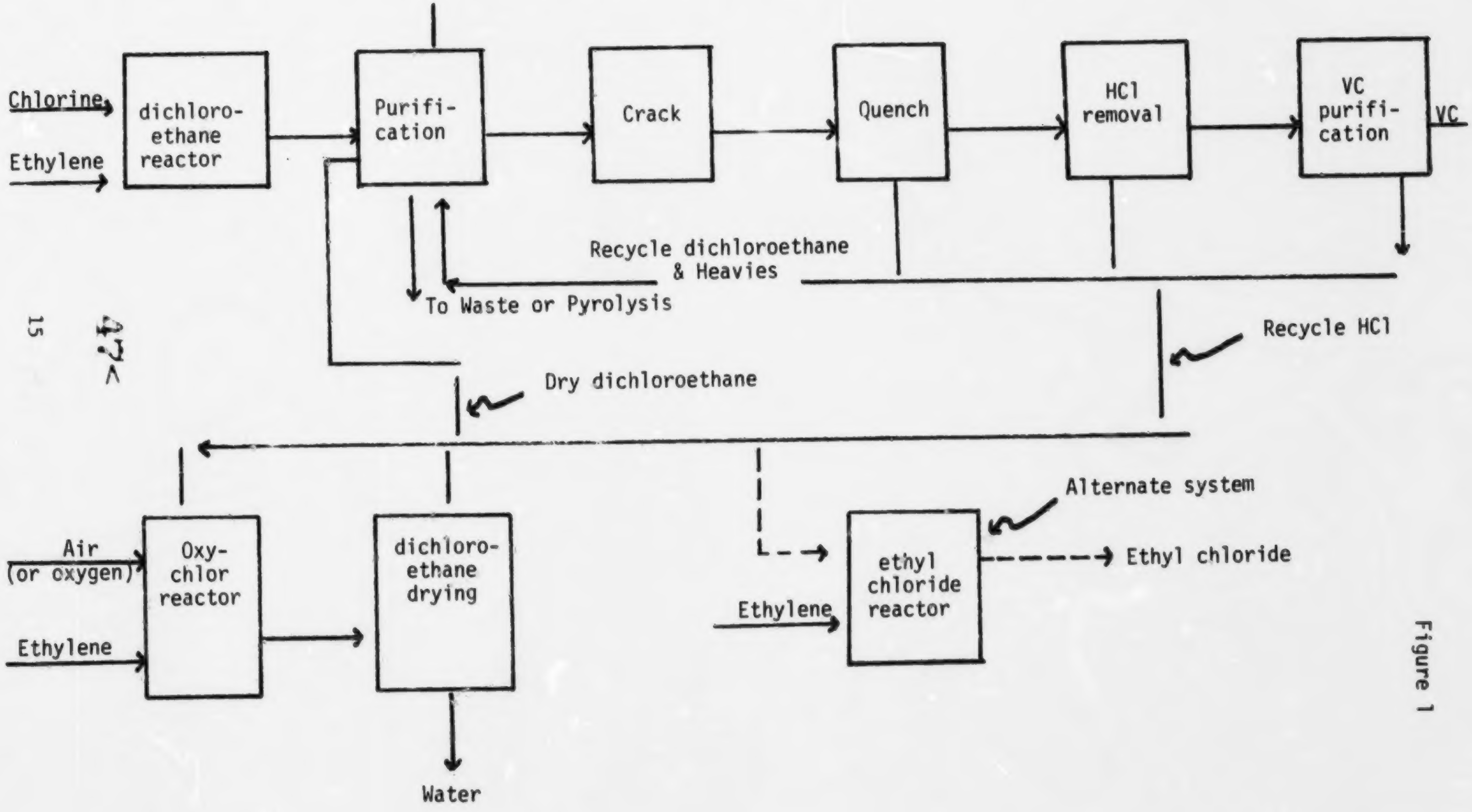
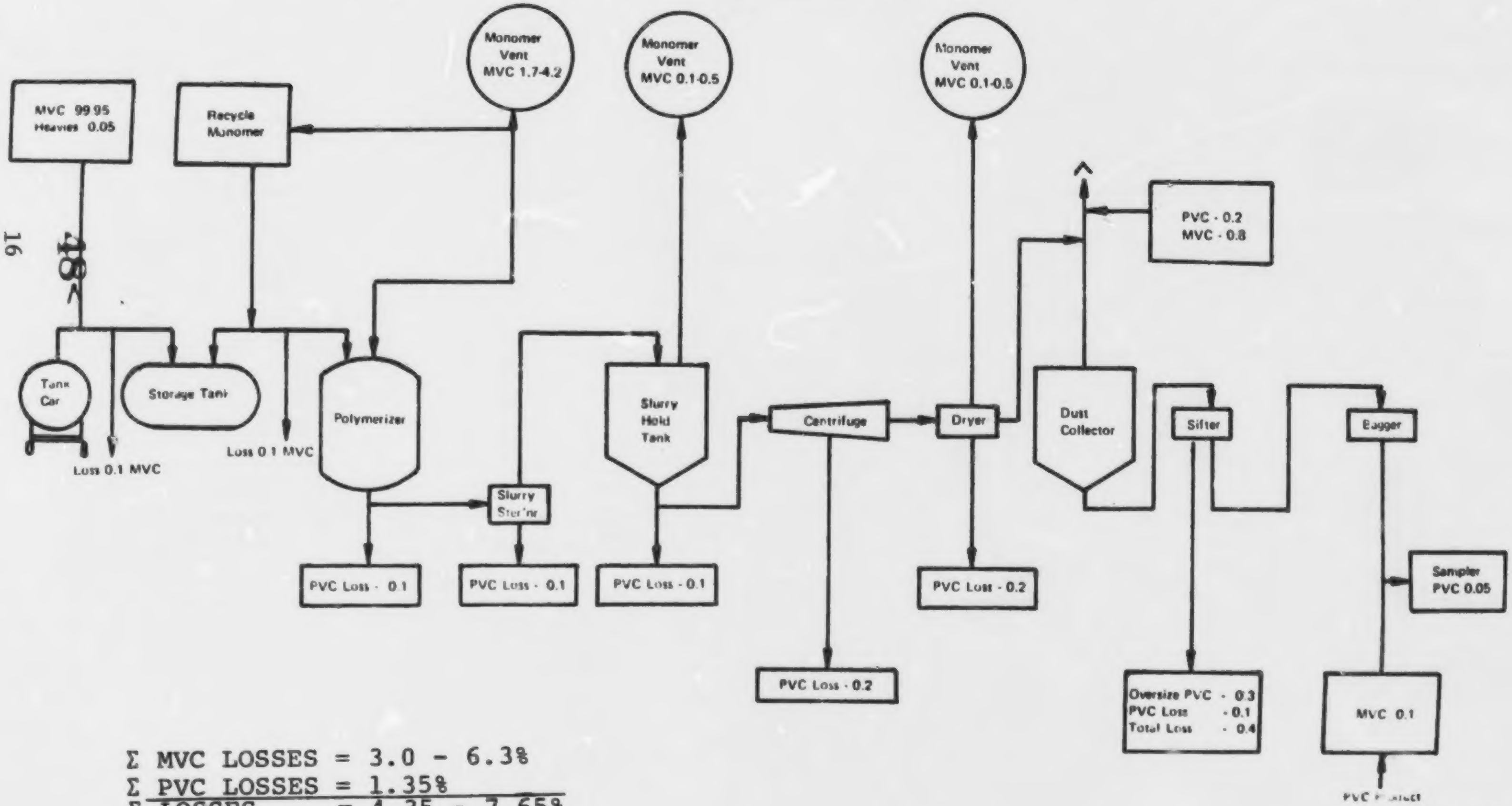

Chlorine
Ethylene
dichloro-ethane reactor
Purifi-cation
Crack
Quench
HCl removal
VC purifi-cation
VC
Recycle dichloroethane & Heavies
To Waste or Pyrolysis
Recycle HCl
Dry dichloroethane
Air (or oxygen)
Ethylene
Oxy-chlor reactor
dichloro-ethane drying
Water
Alternate system
Ethylene
ethyl chloride reactor
Ethyl chloride

Figure 1

47<

Figure 2

PRELIMINARY ESTIMATE OF LOSSES
IN PVC SUSPENSION POLYMERIZATION
(TYPICAL PROCESS)


MVC 99.95
Heavies 0.05
Recycle Monomer
Monomer Vent MVC 1.7-4.2
Monomer Vent MVC 0.1-0.5
Monomer Vent MVC 0.1-0.5
PVC - 0.2
MVC - 0.8
Tank Car
Storage Tank
Loss 0.1 MVC
Loss 0.1 MVC
Polymerizer
Slurry Strainer
Slurry Hold Tank
Centrifuge
Dryer
Dust Collector
Sifter
Bagger
PVC Loss - 0.1
PVC Loss - 0.1
PVC Loss - 0.1
PVC Loss - 0.2
PVC Loss - 0.2
Sampler PVC 0.05
Oversize PVC - 0.3
PVC Loss - 0.1
Total Loss - 0.4
MVC 0.1
PVC Product

Σ MVC LOSSES = 3.0 - 6.3%
Σ PVC LOSSES = 1.35%
Σ LOSSES = 4.35 - 7.65%

48<

INTERIM METHOD FOR SAMPLING AND ANALYSIS OF VINYL CHLORIDE IN WASTE WATER EFFLUENTS AND AIR EMISSIONS

Scope and Application

The initial basis for this method was developed during the monitoring program carried out by EPA Region IV in March and April. The techniques used by Region IV provided guidance for the monitoring activities of other Regions, and the experiences of all Regions were then incorporated into this refined version of the original Region IV approach.

This method is applicable to VC determinations in water effluents, sludges and scums, and atmospheric emissions. The limit of detection is approximately 0.06 mg/l in water and 0.06 ppm (v/v) in air samples.

Summary of Analytical Procedures

Water composite samples, air continuous composite bag samples, and air and water grab samples are analyzed without cleanup by gas chromatography (GC). Separations are effected by selection of one of two types of columns depending upon the nature of the sample. Detection is by means of the flame ionization detector (FID). Tetrahydrofuran extracts of sludges and scums are used for injection into the GC. Air continuous samples on activated carbon are extracted with carbon disulfide, and the extract is analyzed by direct injection into the GC.

Calibration curves are developed using gravimetrically prepared calibration solutions, or by using known dilutions of VC in carrier gas.

VC confirmation should be made by mass spectrometric analysis of the GC eluent if possible. Independent confirmation may also be made in the event of extraordinarily high VC concentration samples by using long path Fourier transform IR spectrophotometry. This IR technique requires special equipment and about 20 cubic feet of air samples.

Interferences

Certain volatile hydrocarbons such as neopentane, butadiene, and freon 12 have elution characteristics similar to VC. However, on the GC column substrates specified in these procedures, these have not usually presented problems of resolution of the VC peak. When column substrates other than those specified have been used, impurities from solvents and carbon adsorbents have been found to interfere with the VC elution peak. Under certain conditions a peak is associated with the injection and subsequent withdrawal of the microsyringe into and from the GC septum. These peaks can also give interferences with the VC peak. Withdrawal should be timed to avoid overlap of this peak with the VC peak.

Apparatus and Materials

Gas Chromatograph

Flame Ionization Detector

Recorder - any potentiometric strip chart recorder which is compatible with the detector system. An integrator is also desirable to estimate peak areas.

Column Materials for Waste Water, Sludge, or Scum Samples

Borosilicate glass tube or stainless steel tube - 6' x 2.5 mm ID preferred. When GC configuration requires columns of other dimensions, these should be used.

Solid support - 60 to 80 mesh Gas Chrom Q

Liquid Phase - 4% FFAP on specified solid support (weight percent). Liquid phase on solid support can be purchased directly from commercial distributors.

Column Materials for Air Samples

Borosilicate glass tubing or stainless steel tubing - 8' x 2.5 mm ID preferred. When GC configuration requires columns of other dimensions, these should be used.

Solid support - Carbopak A

Liquid phase - 0.4% Carbowax 1500 on solid support (weight percent). Liquid support on solid phase can be purchased directly from commercial distributors.

Continuous Air Monitoring Materials - Carbon Adsorption Option

Adsorption Tube - pyrex glass, 18" x 3/8" OD

Activated coconut charcoal, 8-16 mesh. Any good commercial grade, e.g. Fischer Scientific Company can be used.

Becton-Dickson 27 gage 3/8" hypodermic needle flow control

Vacuum pump

Air flow meter

50<

Continuous Air Monitoring Materials and Equipment - Bag Sampling Option

Environmental Measurements, Inc., Programmable Bag Sampler

Tedlar bags (or equivalent)

Gas Pressure Regulator (0-5 PSIG)

Microsyringes - 10, 25, 50, and 100 microliter (graduated)

Gas-tight sample syringes - 1 and 50 ml (graduated)

Vacuum Sampling Cans - 370 ml steel Vacu-Samplers, or glass sampling bottles. Cans and bottles should be flushed with clean air or nitrogen and evacuated prior to use. Evacuated containers should be protected from rough handling to prevent implosion or collapse.

Sampling Bags (Tedlar or equivalent) - 12" x 12", 36" x 36", equipped with sampling valves and speta for GC sample withdrawal

Automatic water sampler - compositor (manual sampling is optional) equipped with sample refrigeration capabilities, and a a means to prevent loss of vinyl chloride from open bottles

Glass sampling bottles with teflon lined screw type caps - 50 ml capacity or other sizes depending upon sampler requirements

Septum-sealed vials - 1 to 10 ml capacity

Volumetric Flask, Glass stoppered, 25 ml

Medicine droppers

Dedicated GC/M.S. for confirmatory tests (preferable)

Barometer

Thermometer

Anemometer

Reagents, Solvents, and Standards

Carrier gases - zero nitrogen or helium

FID gases - zero hydrogen, oxygen

Tetrahydrofuran, reagent grade, peroxide-free

51<

Carbon tetrachloride (reagent grade)

Carbon disulfide (reagent grade)

Standards

VC in zero air, 50 ppm (± 2%) v/v

VC, analyzed reagent grade (lecture bottle)

Sampling

A. Water Samples

All waste water discharge points identified in NPDES permits should be sampled for VC. A minimum of three successive 24-hour composite samples of each site should be taken. Compositing interval should be one hour (manual or automatic sampling is optional). Compositing interval of 20 minutes may be used if the automatic sampler has this capability. Samples should be taken at waste treatment units such as clarifiers and scum and sludge separators. Two 8-hour composites should be taken from the effluents from each of these points, and one 8-hour composite should be taken of scum and sludge from each separator unit.

Compositing interval should be one hour. Three grab samples of clean process water (city or private well) should be taken as blanks.

Samples should be taken in 50 ml bottles with gas-tight, teflon-sealed, screw cap closures, or in equivalent containers required by the characteristics of automatic samplers. All water, sludge, and scum samples should be refrigerated during collection and storage. Compositing volumes should be selected to assure head space above the sample is absent or minimized to avoid loss of VC by its partitioning into the gas phase when samples are sealed. Provisions should be made to avoid such losses during continuous monitoring operations.

Estimates of discharge flows should be made using any appropriate measuring device (venturi, weir, magnetic meter, etc.).

Samples should be preserved by refrigeration and protected from sunlight until they are ready for analysis.

B. Air Samples

Sampling sites should be selected which are downwind and in the plume of the atmospheric emissions from the plant. Samples should be collected only in areas where local residents or neighboring industries would be exposed. At a minimum, sampling should be conducted over a period of five days. Sites should be selected in the following array: one site immediately upwind (A) and one immediately downwind (B) of the plant site; four sites about 0.4 miles from the plant site, one laterally left (C) and one laterally right (D) of the plant site on a line roughly perpendicular to the prevailing wind direction and two (E, F) downwind from the plant site; two sampling sites (G, H) approximately 0.5 miles downwind; single sampling sites, each at distances approximately 0.6 (I), 0.8 (J), 1.0 (K), and 3.0 (L) miles downwind from the plant site. If wind is fish-tailing severely, move sampling sites G and H approximately 0.5 mile upwind of the fish-tailing wind direction from the plant. The sites specified are minimum. Additional sites may be selected contingent on overriding micrometeorological considerations. These should be determined in consultation with the Regional meteorologist. These may be at ground or some elevated level, as determined by the plume survey or as estimated by release of meteorological balloons, anemometer, and wind direction indicators, etc.

SAMPLING SITES

	Prevailing Wind Direction	Minimum Sampling Schedule			
Miles from plant site	Site Symbol	Time	Mon	Wed	Fri
0.0	A				
0.4	C Plant D	0800	A, A, B	A, B, B	A, A, B
0.0	B	1000	C, D, F	C, D	C, D, D
0.4	E F	1200	A, E	A, G, G	A, E
0.5	G H	1400	B, B, F	B, H	B, B, G
0.6	I	1600	C, G	E, K	I, J
0.8	J	1800	D, I	-	L, L
1.0	K	2000	-	H, L, L	-
3.0	L				

(Note: All times are ± 30 minutes for manual grab samples, or ± 2 minutes for automatic, programmable bag samplers).

Grab samples should be taken in 50 ml gas-tight syringes, 50 to 100 ml glass sampling bottles, 370 ml "Vacu-Sampler" metal cans, or 12" x 12" capacity Tedlar-type bags. Both the Vacu-Samplers and the glass sampling bottles should be evacuated prior to use. (Caution: These may implode or collapse when under vacuum. Use due care in their handling). The perfect gas laws should be assumed to estimate gas volumes. Gas-tight syringes are flushed several times with ambient air before a sample is taken. After the sample is taken, the gas-tight syringe is locked and sealed until it is ready for analysis.

The Tedlar-type bag samplers may be filled by pulling the walls of the bag apart manually, or better, by placing the bag in an enclosure and pulling a vacuum on the outside surfaces of the bag. The bag is sealed until it is ready to be analyzed. Tedlar-type bags are preferred for grab sampling.

All samples should be protected from sunlight.

Continuous Sampling - Carbon Adsorption Option:

Continuous samples are taken in pyrex tubes (approximately 3/8" O.D. x 18" long) packed with a good grade of activated coconut shell charcoal. The charcoal is added to the tube in three segments, each 3-inches long, and each separated by a glass wool plug. The two ends of the tube are also plugged with glass wool. Both ends of the pack adsorption tube are plugged with serum caps during transport and for storage purposes.

Flow rate through the tube is controlled by inserting a Becton-Dickson 27 gage, 3/8" hypodermic needle through one of the serum caps into the end glass wool plug. Air is sucked through the tube by connecting it to a conventional vacuum pump. The arrangement is similar to that used in the National Air Surveillance Network. Flow rate should be about 200 ml per minute. For each adsorption tube, the flow rate should be calibrated in the laboratory before the sample is taken and should be verified again in the laboratory after the sample is taken. Clean needles frequently to prevent plugging.

The adsorption efficiency of the carbon in the adsorption tube should be verified in the laboratory by preparing a 5 ppm v/v VC mixture in the 36" x 36" Tedlar-type bag and drawing this through the adsorption tube. Flow rates should be verified before and after the experiment. It is important to note that all collections should be made with the adsorption tubes held in an upright position to minimize channeling. Adsorption tubes should be protected from sunlight either by wrapping with foil or by enclosing them in a box.

Each segment of the adsorption tube is worked up separately by etching the tube in the middle of a 3" section with a file, successively breaking each segment and spilling its contents into measured volumes of carbon disulfide in glass stoppered test tubes. The additions should be effected cautiously and with cooling in an ice bath since the interaction of activated carbon with carbon disulfide is quite exothermic. A 2 microliter aliquot of the supernatant solution should be injected on the carbowax 1500 column for estimation of the adsorped VC. Successive analysis of the three adsorption tube segments will indicate the amount of break-through of VC through the adsorbent.

54<

The same procedure should be used for taking samples in the field.

Continuous Sampling - Programmable Bag Sampler Option: The sampler is programmed to take twenty-four consecutive one-hour composite samples. Each one-hour sample is analyzed separately for VC content. Sampling rate of the individual pumps should be verified before and after use of the sampling device. Record the temperature and atmospheric pressure at which the samples are taken. All gas volumes and concentrations should be corrected to 25°C and one atmosphere (760 mm Hg). At a minimum, continuous samples should be taken at sites A, B, C, and D at ground level, unless otherwise indicated by micrometeorological conditions.

Calibration

A. Gas Analysis - Gas Dilution Option:

Record ambient temperature and atmospheric pressure.

Evaluate the 36" x 36" Tedlar-type bag. Add 1 liter of the standard VC gas mixture (50 ppm, v/v) to the bag. This addition may be made with a flow meter or with a gas-tight syringe. Dilute with nine liters of zero nitrogen or helium carrier gas. This gives a concentration of 5.0 ppm (v/v) of VC. (13 ng/ml at 25°C and one atmosphere.)

Evacuate a 12" x 12" Tedlar-type bag and add 0.5 1 of the 5.0 ppm (v/v) concentration mixture. Dilute with 2 liters of zero nitrogen or helium carrier gas. This gives a concentration of 1.0 ppm (v/v) VC, (2.6 ng/ml at 25°C and one atmosphere).

Evaucate a 12" x 12" Tedlar-type bag and add 0.5 1 of the 1.0 ppm (v/v) VC calibration mixture. Dilute with 2 liters of zero nitrogen or helium carrier gas. This gives a concentration of 0.2 ppm (v/v) VC (about 0.52 ng/ml at 25°C and one atmosphere).

Evacuate a 12"x 12" Tedlar-type bag and add 0.75 1 of the 0.2 ppm (v/v) VC calibration mixture. Dilute with 1.75 liters of zero nitrogen or helium carrier gas. This gives a concentration of 0.06 ppm (v/v) VC (about 0.16 ng/ml at 25°C and one atmosphere). This is about the limit of detection for direct injection into the GC.

With a gas-tight syringe, inject 1 ml aliquots of the 5.0, 1.0, 0.20 and 0.06 ppm (v/v) VC calibration mixtures into a GC equipped with a Carbowax 1500 or Carbopak column and an FID detector. Use zero nitrogen or helium as carrier gas at a flow rate of 60 ml/min. Operate the inlet and the column isothermally at room temperature.

55<

Prepare a calibration curve. Repeat until the calibration curve is reproducible.

B. Gas or Water Analysis - Gravimetric option:

Stock solution of VC.

Pipet 40.0 ml of carbon tetrachloride into a tared 50 ml glass stoppered volumetric flask and accurately weigh to 0.1 mg.

Attach a tygon delivery tube to the VC lecture bottle valve. Attach the end of the delivery tube to a piece of glass tubing which has been constricted at one end, flush out the tube with VC, and slowly bubble VC into the CCl_4 containing volumetric flask until about 5.0 mg of VC has been added. Precautions should be exercised to prevent loss of carbon tetrachloride during this operation. Reweigh the volumetric flask to determine the weight of added VC. Fill the volumetric flask to the 50 ml mark (approximately 100 ppm wt/vol). (These operations should be carried out in a hood).

Transfer 1 ml of the stock solution of VC to a 25 ml volumetric flask and dilute to the 25 ml mark with carbon tetrachloride (approximately 4 ppm w/v).

Transfer 5 ml of the 4 ppm VC solution to a 10 ml volumetric flask and dilute to the 10 ml mark (approximately 2 ppm, w/v). Repeat dilution for a solution approximately 1 ppm, and 0.2 ppm.

Transfer the stock solution to a teflon-lined screw capped bottle. This solution can be kept for extended periods of time Transfer the diluted solutions to serum vials and cap them with teflon-lined serum cap septa.

Inject 1 ml aliquots of the calibration solutions in the GC equipped with Carbowax 1500 on Carbopak A packed columns and an FID detector. Use Zero nitrogen or helium carrier gas at a flow rate of 60 ml/min. Operate the inlet at 150°C and the column at 60°C. After the VC peak has been eluted, program the column temperature to 150° C to elute solvent. Cool column back to 60°C for follow-on concentrations.

Repeat procedure using a GC equipped with a 4% FFAP on Gas Chrom Q packed column and FID detector. Operate under the same conditions. Prepare a calibration curve to be used be used with water samples.

Procedure

Water Sample Analysis

Untreated water samples (1-5 microliter aliquots) are injected directly into the GC.

A 4% FFAP on "Gas Chrom Q" packed column is used. Nitrogen zero gas or helium is used as the carrier gas at a flow rate of 60 ml/min. Inlet temperature is set at 150°C. The column is operated isothermally at 62°C. Detection is by FID.

Report concentration of VC in sample in mg/l.

Sludge and Scum Samples

Extract 5 grams of sludge or scum sample with 100 ml of tetrahydrofuran (THF). Analyze THF extract in the same manner used for water samples. If VC concentrations are too high, make appropriate dilutions of the THF extracts.

Report concentration of VC in sample in mg /g of sample.

Air Sample Analysis

Grab samples.

Use a 0.4% Carbowax 1500 on Carbopak A packed column. Use nitrogen zero gas or helium as the carrier gas with a flow rate of 60 ml/min. Operate the column and inlet at room temperature. Use a flame ionization detector.

Untreated air samples (1 ml) are injected directly into the GC. VC contamination of syringes requires attention.

Report concentration of VC in gas samples in ppm (v/v).

Continuous Samples

Use same procedure as previously discussed for calibration of adsorption tube efficiency.

Quality Control

Duplicate sample analyses are recommended as a quality control check.

57<

APPENDIX V

SUMMARY OF REGIONAL ACTIVITIES

This Appendix briefly summarizes the results of the preliminary VC monitoring activities conducted by EPA Regional Offices during the Spring of 1974 at the request of the Task Force. More detailed reports are available from the Regional Offices.

The sampling and analyses were carried out in a very short period of time using new methods, based on the Agency's best scientific judgement. They represent, in the Agency's opinion, the best methods then available. In large measure, the sampling and analysis methods were based on previous analytical studies in which similar chemicals were evaluated. However, they had not been thoroughly tested for accuracy and precision under field conditions.

Prior to and during the sampling and measurement only limited quality control and standardization of procedures could be applied in the time available. The methods utilized were interim procedures which have already been subjected to further modification.

The nature of the PVC manufacturing process results in the escape of VC pulses which could lead to widely fluctuating levels of VC in the ambient air. So, too, changes in air movement may influence concentrations at a given station at any one time. Therefore, the VC data reported are preliminary in nature and are subject to change as additional monitoring is performed. Individual measurements probably underestimate the VC levels due to the possibility of VC leakages and other inaccuracies in the monitoring system.

Region I: Leominster, Massachusetts: Borden Chemical Company (PVC); May 9, 10, 13.

1. One hundred and fifty-seven discrete (grab) ambient air samples were collected on plant property and within a 3.0 mile radius of the plant. The VC concentrations ranged from less than the detectable limit of 0.06 ppm to 6.0 ppm. The samples exceeding 1 ppm were obtained on plant property near the fenceline.

2. Twelve 24-hour integrated ambient air samples were collected at the fenceline on plant property. The VC values ranged from less than the detectable limit of 0.06 ppm to 1 ppm.

3. VC concentrations in three 24-hour composite waste water samples taken from the lagoon effluent ranged from 0.15 to 0.29 ppm.

4. VC concentrations in two sludge samples taken from the lagoon near the outlet measured at the 0.05 - 0.06 ppm level on a wet basis.

5. The plant is located in a residential/industrial area on the edge of Leominster with residential developments adjacent to plant property.

6. Shifting meteorological conditions and rain hampered the sampling program.

Region II: Flemington, New Jersey: Tenneco Chemicals, Inc. (PVC); May 29-31.

1. Forty-three discrete ambient air samples were collected on plant property and within a 2.0 mile radius of the plant. The VC concentrations outside the plant property ranged from less than detectable (0.01 ppm) to 0.05 ppm. On plant property a single sample collected on the dryer building roof contained 5.6 ppm. At ground elevation, the VC concentrations on plant property ranged up to 0.30 ppm.

2. Twenty-three integrated ambient air samples were collected for 24-hour periods on plant property and within 2.0 miles of the plant. The VC values ranged from 0.005 to 0.038 ppm on plant property and from less than detectable to 0.031 ppm outside the plant area.

3. Two integrated one-hour ambient air samples collected within 0.1 mile of the plant showed VC at levels of 0.32 ppm and 0.18 ppm.

4. A maximum level of 20 ppm was detected in three 24-hour composite samples taken from the water effluent discharge into the Bushkill Brook, which immediately flows into the Raritan River. This amounts to approximately 400 lbs/day.

5. VC concentrations in sludge samples taken from the lagoon areas on plant property ranged from less than detectable to 1,000 ppm in wet weight concentrations; however, the concentration at the sludge disposal area was 54 ppm.

6. The plant is located in an area in which manufacturing facilities are interspersed with farmland and relatively large acreage residential properties. There are a number of small communities within a few miles of the plant.

Region III: Delaware City, Delaware: Stauffer Chemical Company (PVC) and Diamond Shamrock Chemical Company (PVC); May 20-22. S. Charleston, West Virginia: Union Carbide Corporation (PVC); May 24.

1. The air sampling and analysis activity was organized around a mobile laboratory equipped with a gas chromatograph using a flame ionization detector. VC levels were later confirmed by mass spectrometer.

2. A single discrete ambient air sample at the fenceline of the Diamond Shamrock plant showed 0.2 ppm VC.

3. Four discrete ambient air samples taken near the Stauffer Chemical plant ranged from 0.3 to 0.7 ppm VC. The highest level was recorded 0.5 miles from the plant and the lower levels at 0.25 miles from the plant.

4. The area immediately adjacent to the Delaware City complex is lightly populated residential areas for several miles.

5. Water samples collected at the Union Carbide plant gave VC values of 1.1 and 0.8 ppm for grab samples at several outfalls and 0.35 for a 24-hour composite. Samples obtained from the Kanawha River did not have a detectable level of VC.

6. Sampling was attempted but was not feasible due to limited time and equipment difficulties at the PVC plants of the Firestone Plastics Company in Perryville, Maryland, and Pottstown, Pennsylvania.

Region IV: Louisville, Kentucky: B.F. Goodrich Chemical Company (PVC); March 19-21 and May 8-16.

1. The initial air monitoring program conducted in March was preliminary to the more extensive program in May which showed significantly higher levels.

2. In May there were 39 discrete ambient air samples collected in the area designated industrial (within 0.8 miles from the plant center). The VC concentrations ranged from less than 0.05 to 5.6 ppm, with 10 samples exceeding 1 ppm. In the area designated residential/ industrial, 149 samples were collected within 0.8 miles of the plant with VC concentrations ranging from less than 0.05 to 33 ppm. The average concentrations at the site registering 33 ppm were between 0.5 and 1 ppm, but 18 samples had concentrations greater than 5.0 ppm. Four samples were obtained in strictly residential areas with VC values of 0.05 to 1.6 being observed. The 1.6 value was 0.8 miles from the plant.

3. Five sampling sites were established within 0.6 miles of the plant for integrated air sampling over 24 hours. VC values ranged from less than 0.001 to 0.53 ppm. The highest value was obtained from a sampling site 0.2 miles from the plant center.

4. Wastewater from the clarifier discharge was measured in March at 2 to 3 mg/l in 24-hour composite samples.

5. Dewatered clarifier sludge and clarifier scum contained 193 and 162 ppm of VC, respectively.

Region V: Painesville, Ohio: Uniroyal, Inc. (PVC) and Robintech, Inc. Inc. (PVC); May 9-14.

1. Four of 137 ambient air samples taken at distances up to 3.0 miles from the plant showed levels exceeding 1 ppm of VC with the highest level being 2.26 ppm. Many of the samples were less than 0.1 ppm.

2. Nine 24-hour integrated ambient air samples taken at various distances from the plant showed levels up to 0.2 ppm of VC.

3. VC levels in 11 of 17 water effluent samples were less than 0.2 ppm, with three samples exceeding 1 ppm, including a high of 3.7 ppm.

4. VC levels in nine sludge samples, as the sludge would leave the plant property, ranged from 9 to 3520 ppm.

5. The complex is surrounded by residential areas.

Region VI: Plaquemine, Louisiana: The Goodyear Tire and Rubber Company (PVC) and Dow Chemical Company (VC); April 7-9.

1. There were 31 discrete ambient air samples collected within 3.0 miles of the complex with VC concentrations ranging from less than detectable (.001 ppm) to 7.81 ppm. Most of the readings were less than 1 ppm, with the highest value at the property line.

2. VC concentrations in wastewater effluent measured by 24-hour composites were all below .05 ppm.

3. VC concentrations in residual reactor scrapings at the Goodyear plant ranged from 23 to 31 ppm.

4. The small communities of Morrisonville and Eliza are located less than 1 mile north and northwest respectively of the Goodyear plant. A few homes from Morrisonville extend almost to the north property line of the Goodyear plant.

5. Very limited air sampling was conducted in the Houston area in the vicinity of the plants listed below. However, in view of the inadequacy of this activity, the sampling effort in this area is being continued.

Deer Park, Tex., PVC Plant - Diamond Shamrock Corp., Diamond Shamrock Chemical Co.

Deer Park, Tex., VC Plant - Shell Chemical Co., Industrial Chemicals Division

Houston, Tex., VC Plant - Tenneco, Inc., Tenneco Chemicals, Inc.

Pasadena, Tex., VC Plant - Ethyl Corporation

Region IX: Long Beach, California: B.F. Goodrich Chemical Company (PVC); American Chemical Corporation (VC); American Chemical Corporation (PVC); May 7-10.

1. One hundred and eighty 10-minute integrated ambient air samples were collected within 3.1 miles of the complex. About 11 percent of the

readings exceeded 0.5 ppm, while 5 percent exceeded 1.0 ppm. The maximum value measured was 3.4 ppm in a sample taken 3.1 miles from the plant; however, the average level measured at this point was about 0.5 ppm.

2. Samples of wastewater effluents were composited for 8 to 24 hours and yielded values from 3.5 to 8.9 ppm, with individual samples reading up to 22 ppm.

3. Sludge samples showed values ranging from 290 to 4200 micrograms of VC per gram of dry sludge.

4. The complex is surrounded by residential areas. Within the three mile radius of the plants there are eleven schools.

62<

APPENDIX VI

PERSISTENCE OF VINYL CHLORIDE

The available information on the stability and persistence of VC in the environment is currently very limited. Some literature and laboratory studies have recently been initiated by industry and by EPA. This discussion summarizes the findings of EPA to date and particularly the results of research efforts at EPA research facilities undertaken in response to the needs of the Task Force for at least preliminary data on environmental fate. Results of related experiments reported by industry seem to be consistent with the discussion.

Behavior of Vinyl Chloride in Air

The peak absorption of VC in the ultraviolet region is very far below the solar cutoff of about 2900 A, indicating that VC would not undergo reaction in sunlight in the absence of other reactive chemicals. When irradiated with simulated solar radiation in the presence of nitrogen oxides (nitric oxide and nitrogen dioxide), VC reacts to form a variety of products. The available laboratory results indicate a rate of reaction of about 8 to 10% per hour for VC, recognizing that reaction rates may vary with concentrations. The direct and indirect reaction products identified included ozone, nitrogen dioxide, carbon monoxide, formaldehyde, formic acid, and formyl chloride. High eye irritation levels were found with human exposure panels which is consistent with the products identified.

The low reaction rate of VC, including reactions in the presence of nitrogen oxides, indicates that within a few miles downwind of VC emission sources VC will persist and can be considered a stable pollutant. The usual meteorological dispersion equations for gases could be applied to approximate concentrations. Because of temperature inversions and the absence of sunlight at night during the fall and winter, buildup of VC might be of particular concern during such periods. Clearly at greater distances from emission sources, VC will have greater opportunity to disperse and degrade.

The noxious gases which are products of VC reactions should not be ignored. In air quality regions with large industrial activities involving large volume production of these chemicals, such products may contribute appreciably on particularly sunny days to eye, nose, throat, and lung irritation.

Behavior of Vinyl Chloride in Water

The loss of VC from water at constant temperature and pressure depends on the rate of agitation or aeration. Distilled water in a beaker spiked with 16 ppm VC, when rapidly stirred at 22°C with a magnetic stirrer, lost 96% of VC in two hours, while quiescent water at the same concentration lost only 25% VC. There was no significant difference in the rate of VC losses from distilled water, river water, or effluent from a VC plant stirred at the same rate, indicating negligible adsorption effects with particulate matter. Plots of log water concentration versus time give straight lines, indicating volatility to be the only important loss mechanism.

63<

Hydrolysis over a pH range of 4.3 to 9.4 does not appear to be an important pathway for loss of VC from water. Chemical reaction of VC in the clarifier effluent from a VC plant was followed at 50°C for 57 hours at pH 4.3, 8.0, and 9.4 in sealed septum vials. Concentrations indicated that VC at these three pH values decreased at the same rate. This lack of pH dependence suggests that the loss of VC occurred by volatilization rather than hydrolysis, or at least there is a very slow hydrolysis rate. This experiment should be repeated in leak-proof reaction vials.

Very preliminary experiments do not show photolysis as an important pathway for loss of VC in water. However, there are many uncertainties in the experimental techniques, and additional studies are needed in this area.

Earlier theoretical studies are consistent with these experimental results. One study on the transfer of small non-reactive molecules across the air-water interface (as in stream aeration) used a kinetic approach to predict that VC will be rapidly lost from an aqueous solution, with the rate of loss being a function of water turbulence, mixing efficiency, and molecular diameter. Another study, using a thermodynamic approach, predicted a rapid rate of evaporation of low solubility chlorinated hydrocarbons, including compounds of low vapor pressure.

Despite the foregoing efforts there is a general absence of data concerning VC in aquatic systems. It is conceivable that as the result of poor or erratic mixing in lakes or ponds, together with slow but continuous release of VC from sediments and sludges, VC could persist long enough to accumulate biologically, via direct absorption or via the food chain, or to cause other ecological effects.

Behavior of Vinyl Chloride in Closed Rooms

Tables 1 and 2 present data concerning concentrations of VC in a typical room following release of a pesticidal spray containing VC.

TABLE 1
One Hundred and Twenty Second Release of Insect Spray in 133,000 Liter Room

		COLUMN I		COLUMN II	
SAMPLE	TIME	VC	FREON-12	VC	FREON-12
No. 1	Collected at breathing zone during spray	41.64 ppm	8.15 ppm	41.9 ppm	7.94 ppm
No. 2	15 minutes	16.91	3.13	17.1	3.30
No. 3	30 minutes	1.38	0.27	1.32	0.25
No. 4	60 minutes	0.08	0.018	0.061	0.018
No. 5	120 minutes	0.012	-	0.010	-

64<

TABLE II

Thirty Second Release of Insect Spray in 21,400 Liter Room

		COLUMN I		COLUMN II	
SAMPLE	TIME	VC	FREON-12*	VC	FREON-12*
No. 1	Collected one minute after spray	380.1 ppm	84.8 ppm	383.6 ppm	83.2 ppm
No. 2	30 minutes later	52.1	9.9	48.7	10.3
No. 3	60 minutes	24.6	4.8	22.5	4.7
No. 4	150 minutes	10.3	2.1	9.3	2.2
No. 5	Collected in adjacent hall 151 minutes	0.83	0.17	0.17	0.15

*Freon-12 concentrations were determined using hydrocarbon response factors to compare dilution effects; the actual concentration is higher by a factor of 5.3.

REFERENCES

1. Unpublished results of experiments and analyses conducted at EPA laboratories in Research Triangle Park, N. C., and Athens, Georgia, during April and May 1974.

2. Unpublished results of experiments on persistence of VC in water conducted by Dow Chemical Company.

3. Tsiroglou, E. C. and J. R. Wallace, "Characterization of Stream Reaeration Capacity," EPA Ecological Research Series Report #EPA-R3-72-012 (October, 1972).

4. MacKay, Donald and Aaron W. Wolkoff, "Rate of Evaporation of Low-Solubility Contaminants from Water Bodies to Atmosphere," Environmental Science & Technology, 7 (7):611-614 (July, 1973).

65<

HEALTH EFFECTS OF VC

This Appendix presents much of the epidemiological and toxicological data available as of August 1974, on the health effects associated with exposure to VC, together with a few interpretive comments supplementing information presented in the body of the report. However, the Appendix does not present an exhaustive review or evaluation of available information.

Table 1 summarizes the data, collected by CDC/NIOSH, on the confirmed cases of angiosarcoma of the liver in VC/PVC workers in the United States and abroad. A total of 15 occupational cases have been discovered in the United States and confirmed as angiosarcoma of the liver. Of the 15 cases, 2 are still alive and undergoing treatment. Fourteen of the 15 were employed in PVC production plants and the remaining one in a PVC fabrication plant. The average age at death for the U.S. PVC production workers was 48.5 years (with a range from 36 to 61 years) which is about seven years younger than the average age of death from liver cancer in the U.S. male population. Based on the data available for the workers, the latent period for this disease appears to be on the order of twenty years, a period consistent with latencies observed for other occupational, chemically induced cancers.

In the U.S. PVC production worker cases, all of the men were at one time "pot cleaners", required to enter the reactors in order to chip the residue of the chemical reaction from the sides of the "pots." Since the residue often contained pockets of trapped gases that were literally released in the cleaner's face when they were ruptured by his chipping operation, the potential for exposure to high levels of VC while cleaning these tanks was particularly great during the early years of this operation.

Ten cases of worker-related angiosarcoma of the liver have been reported from five foreign countries to date.

Table 2 summarizes the epidemiological data, collected by CDC from the Connecticut Tumor Registry, on five confirmed cases of angiosarcoma of the liver, including one accountant in a PVC fabrication plant and two residents near PVC fabrication plants. The case of occupational exposure occurred in a man who had been employed for 10 years as an accountant in a factory which produces vinyl sheets and processes PVC resins; it is reported that he frequently visited the production area of the plant. Of the two cases who had no occupational exposure to VC or PVC, one was a 73 year-old man who lived his entire life within two miles of a PVC wire insulation plant. The other was an 83 year-old woman, a housewife and retired cook, who had lived for 35 years within one-half mile of the vinyl products plant at which the accountant had been employed.

While these findings establish no causal connection between exposure to PVC and angiosarcoma of the liver, they do raise the possibility of such a relationship. Time will be needed to define the possible risk factors in persons who have worked with PVC since the latency period appears to be so long. Because of the rarity of this tumor, the additional finding in this study of angiosarcoma of the liver in persons who had no occupational exposure to VC, but who may have had community exposure, is also worrisome but again establishes no causal connection. Epidemiologic investigation of additional cases of hepatic angiosarcoma that may be found to have had possible community exposure to VC will be necessary to clarify the significance of these cases.

Tables 3A - 3D present the findings of the MCA-funded mortality study of VC/PVC workers, conducted by Tabershaw/Cooper Associates.

In calculating the risk of death, the usual method is to express the number of deaths which actually occurred as a percentage of the number which would have been expected in a comparable population observed over the same age and time intervals. This statistic is called the Standardized Mortality Ratio (SMR). Using the U.S. male population as the standard population of comparison, the SMRs were calculated for each of the 35 cases of death for which detailed mortaility rates are published on a national basis. In the standard population each SMR would be equal to 100. The statistical significance of the deviation of each SMR in the study population from the expected value of 100 was tested. A single asterisk indicates those SMRs which differed significantly from 100 at the 5 percent level, that is, which had a probability of .05 or less of occurring by chance. A double asterisk indicates those which were significant at the 1 percent level. SMRs based on fewer than 5 observed cases were not tested for significance. The overall mortality of the study population is statistically significantly lower than that of the U.S. male population. There were 352 observed deaths compared with 467 expected, for an SMR of 75.

For each job, an exposure score was estimated by industrial hygiene and safety personnel in each plant. A score of 1 was given for low exposure, 2 for medium, and 3 for high. The number of months each worker spent on a given job was multiplied by the appropriate exposure score. The total for each worker was then divided by the total number of months of exposure to give an Exposure Index (EI) for that worker. Table 3A shows the SMRs for workers with an EI below 1.5 versus those at 1.5 or above. The dividing point of 1.5 represents a level halfway between low and medium exposure. Table 3B shows similar results for workers with less than 5 years exposure versus those with 5 years or more.

In order to examine the possible interaction between duration and level of exposure, the study population was divided into 4 groups on the basis of both EI (low vs. high) and duration of exposure (short vs.

long) using the same dichotomization as Tables 3A and 3B. Table 3C shows the results for short versus long exposure in the low El group, and Table 3A shows the same comparison in the high EI group. When the study population is divided according to length and duration of exposure (Tables 3A and 3B) and combinations of these measurements (Tables 3C and 3D), three major patterns emerge. For malignant neoplasms as a whole, the SMR increases with increasing exposure, whether measured by level, duration, or both. In the high exposure group with 5 years or more exposure (Table 3D) there are 36 observed cases and 26.11 expected. For cardiovascular - renal diseases as a group, there are also increases in the SMR with increasing exposure, but the number of observed cases remain less than expected, the differences being statistically significant in all groups except the high exposure, long duration group. For all other causes, there are no consistent relationships with exposure.

Within the malignant neoplasms, the largest (although not statistically significant) SMR is in cancers of the buccal cavity and pharynx, with 5 observed, 2.64 expected, and an SMR of 189. However, Tables 3A and 3D show that all these cases have an EI below 1.5, and 4 out of 5 have less than 5 years exposure.

Cancer of the digestive system shows no excess in the study population as a whole. However, in those workers with EIs of 1.5 or higher, there are 12 observed cases where 9.14 are expected (Table 3A). In the subgroup of the above workers with 5 years or more exposure, there are 11 observed cases and 7.47 expected.

Respiratory cancer shows a slight excess in the total group, and a similar pattern for different exposure categories, with 13 observed versus 10.28 expected when the EI is 1.5 or higher, and 12 observed versus 8.50 expected when, in addition, the duration of exposure is 5 years or more.

Malignant neoplasms of other and unspecified sites show an excess in the total group, and an increase with both level and duration of exposure (Tables 3A and 3B). The relationship with exposure is more pronounced, since those with exposures of less than 5 years have fewer cases than expected.

The lymphosarcomas, although occurring at about the expected rate when the whole group is considered, are concentrated almost entirely in the high exposure long duration group. In that category there are 4 cases observed and 1.84 expected.

The Tabershaw/Cooper Study is based on an examination of 328 death certificates. The authors acknowledge three areas where bias might have entered: (a) choice of the U.S. male population as the standard, (b) absence of 15% of the study population (untraceable), and (c) discovery, as the study ended, of a group of 1500 workers whose exposures occurred up to 35 years ago and who are not included in the study group. Since the latency period for angiosarcoma of the liver is averaging 18 years at least, it would appear desirable to examine the data for these 1500 workers.

In addition to the Tabershaw/Cooper study several other epidemiological studies presented during the recent OSHA hearings suggest the possibility of a multiple cancer risk.

Table 4 summarizes many of the published and unpublished toxicological and epidemiological studies of human and animal exposures to VC. A list of the references cited in Table 4 completes this Appendix.

Table 1
OCCUPATIONAL CASES OF LIVER ANGIOSARCOMA

Occupation	Country	Case #	BIRTH DATE	1st VC/PVC Work	Diagnosis of Angiosarcoma	Age at Diagnosis	Yrs. 1st VC/PVC Work To Diagnosis	Total Yrs. VC/PVC Exposure	Date of Death
1. VC Monomer Production	Sweden	01	00-00-11	00-00-45	00-00-72	61	27	23	00-00-72
2. PVC Polymerization	United States	01	00-00-22	12-09-48	03-00-71	49	22	16	03-03-73
	United States	02	00-00-34	11-15-55	05-00-70	36	14	13	09-28-71
	United States	03	00-00-15	11-28-45	12-00-73	58	28	28	12-19-73
	United States	04	00-00-24	07-06-52	08-00-67	43	15	15	01-07-68
	United States	05	00-00-12	06-19-44	04-00-64	52	20	18	04-09-64
	United States	06	00-00-29	01-17-62	02-00-74	45	12	12	Alive
	United States	07	05-03-22	08-00-44	00-00-68	45	24	18	03-23-68
	United States	08	05-06-20	10-07-46	08-00-61	41	15	15	08-29-61
	United States	09	00-00-31	05-28-45	03-01-74	43	29	17	Alive
	United States	10	08-16-13	06-00-51	05-00-68	55	17	17	05-10-68
	United States	11	05-27-09	10-14-46	03-00-70	61	23	23	03-16-70
	United States	12	11-17-18	09-13-49	05-00-69	50	20	15	05-02-69
	United States	13	12-01-21	08-19-44	05-00-74	53	30	30	07-04-74
	W. Germany	01	07-26-31	10-14-57	00-00-71	40	14	14	12-14-71
	W. Germany	02	06-04-30	10-01-57	00-00-69	39	11	11	01-25-69
	Great Britain	01	00-00-01	00-00-46	12-00-72	71	26	20	12-00-72
	Norway	01	12-23-15	03-00-50	12-20-71	56	22	21	01-04-72
	Sweden	02	00-00-27	00-00-51	00-00-70	43	19	18	00-00-70
	Czechoslovakia	01							
	Czechoslovakia	02							
	United States	14	11-04-27	11-11-51	00-00-69	41	17	4	03-27-69
3. PVC Compounders, Fabricators, Etc.	United States	15	00-00-25	00-00-00	07-00-72	47	00	00	02-15-73
	Great Britain	02	09-09-14	00-00-46	02-00-70	55	24	11	12-00-70
4. Other VC Exposure	W. Germany	03				43	14		

Note: '00' indicates unknown date

SOURCE: NIOSH

70<

Table 2

CASES OF HEPATIC ANGIOSARCOMA, CONNECTICUT, 1935-1973

Case No.	Age	Sex	NCI Diagnosis	Date of Original Diagnosis	Date of Death	Medical History	Occupation	Place of Residence
1	73	M	Hepatic Angiosarcoma	11-25-67	12-3-67	2 months history of diarrhea, , anorexia, and 20 lb weight loss. Intermittent abdominal pain. Non-tender, firm epigastric mass. Died after 9 days with spontaneous ruptured liver leading to shock. Past history of alcohol intake.	Fireman 1917-42 Aluminum worker 1942-44 Corset cutter 1945-61 Retired 1961-67	Bridgeport - entire life
2	47	M	Alcoholic Cirrhosis Portal Fibrosis	1-15-73	2-15-73	Initial symptoms RUQ abdominal pain with vomiting. Cecal volvulus found, Rx cecopexy, over next 6 weeks pain continued with weakness. RUQ tenderness with 2 FB liver. Diagnosed by needle biopsy on 1-15-73. Deteriorated slowly until death 31 days later.	Accountant - Vinyl Co., 1963-73 Accountant - Plastic Belt Co., 1956-63 Previously accountant-other states	Bridgeport - 1956-73 Previously many locations
3	83	F	Hepatic Angiosarcoma	12-19-73	1-22-74	Admitted 12-2-73 with short history of RUQ abdominal pain radiating to R shoulder. Had RUQ tenderness. Open liver biopsy 12-19-73 showed large tumor. No resection. Deteriorated until death 34 days later.	Housewife Restaurant cook 35 yrs	Stratford 35 years
4	76	F	Hepatic Angiosarcoma	3-12-50	3-19-50	1 month history of anorexia with abdominal pain and back pain. Firm epigastric mass. Died 6 days after admission with carcinomatosis and pulmonary emboli.	Housewife	Windsor Locks
5	50	M	Hepatic Angiosarcoma	-	5-4-73	Admitted for abdominal pain and jaundice 3-27-73. 4 FB liver. Discharged. Readmitted 4-29-73 with abd distension, general edema, icterus, fever, shaking chills. Rapid downhill course with death due to renal and hepatic failure. Past history of alcohol intake.	Fisherman and carpenter before 1959 Plasterer 1959-60. Unemployed 1960-73.	Puerto Rico 1923-59 New York City 1959-73 Bridgeport 1973

71<

Table 3A

OBSERVED DEATHS/EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS IN VINYL CHLORIDE WORKERS, BY ESTIMATED LEVEL OF EXPOSURE

Cause of death with I.C.D.+ number	EI <1.5		EI ≥1.5	
	obs/exp	SMR[1]	obs/exp	SMR[1]
All causes	188/270.33	70**	157/195.68	80**
Tuberculosis (001-019)	0/3.38	0	0/2.33	0
Tuberculosis of respiratory system (001-008)	0/3.16	0	0/2.18	0
Malignant neoplasms (140-205)	37/44.28	90	41/32.67	134
Malignant neoplasms, buccal cavity and pharynx (140-148)	5/1.62	330	0/1.21	0
Malignant neoplasms, digestive organs and peritoneum (150-159)	7/12.50	60*	12/9.14	141
Malignant neoplasms, respiratory system (160-164)	11/13.56	86	13/10.28	135
Malignant neoplasms, genital organs (170-179)	2/2.30	93	1/1.43	75
Malignant neoplasms, urinary organs (180-181)	1/2.07	51	0/1.52	0
Malignant neoplasms, other and unspecified sites (190-199)	9/6.57	146	8/4.52	190
Leukemia and aleukemia (204)	1/2.18	49	2/1.57	136
Lymphosarcoma, lymphatic and hematopoietic tissues (200-203, 205)	1/3.48	31	5/2.54	212
Diabetes mellitus (260)	5/3.65	146	2/2.65	81
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	84/120.11	75**	69/86.99	85*
Vascular lesions affecting CNS (330-334)	7/14.42	52**	6/10.06	64
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	3/3.98	80	2/2.85	75
Arteriosclerotic heart disease (420)	68/78.94	92	51/58.05	95
Nonrheumatic endocarditis (421, 422)	0/4.20	0	1/2.89	38
Hypertensive heart disease (440-443)	1/5.48	19	2/3.86	56
Other hypertensive disease (444-447)	1/1.52	70	2/1.07	201
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/2.50	0	0/1.77	0
Influenza and pneumonia (480-493)	5/5.80	92	0/4.13	0
Ulcer of stomach and duodenum (540, 541)	1/2.21	48	1/1.60	68
Appendicitis (550-553)	0/0.39	0	0/0.27	0
Hernia and intestinal obstruction (560, 561, 570)	0/0.88	0	1/0.63	171
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	0/0.76	0	1/0.55	196
Cirrhosis of liver (581)	2/8.90	23	1/6.64	16
Hyperplasia of prostate (610)	0/0.25	0	0/0.14	0
Symptoms, senility and ill-defined conditions (780-795)	0/4.22	0	1/3.09	34
All other diseases (residual)	14/21.90	68*	6/15.89	41**
Motor vehicle accidents (810-835)	8/19.08	45**	9/13.46	72
Other accidents (800-802, 840-962)	11/17.83	66*	6/12.67	50**
Suicide (963, 970-979)	9/9.73	98	7/7.02	107
Homicide (964, 980-985)	0/6.98	0	1/4.94	21
Number of workers	4032		3057	
Person-years	43354		32108	

[1] SMR's adjusted for deaths with cause unknown.

*Significant at 5% level.

**Significant at 1% level.

+International Classification of Diseases

SOURCE: Tabershaw Cooper Associates, Inc., Epidemiological Study of Vinyl Chloride Workers, Final Report

72<

Table 3B

OBSERVED DEATHS/EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS IN VINYL CHLORIDE WORKERS BY DURATION OF EXPOSED EMPLOYMENT

Cause of death with I.C.D. number	<60 months obs/exp	<60 months SMR[1]	≥60 months obs/exp	≥60 months SMR[1]
All causes	94/140.53	67**	251/329.30	76**
Tuberculosis (001-019)	0/2.23	0	0/3.55	0
Tuberculosis of respiratory system (001-008)	0/2.07	0	0/3.33	0
Malignant neoplasms (140-205)	13/19.96	78	65/57.61	116
Malignant neoplasms, buccal cavity and pharynx (140-148)	4/0.70	688	1/2.16	47
Malignant neoplasms, digestive organs and peritoneum (150-159)	2/5.26	46	17/16.56	106
Malignant neoplasms, respiratory system (160-164)	3/5.52	65	21/18.51	116
Malignant neoplasms, genital organs (170-179)	0/0.99	0	3/2.76	112
Malignant neoplasms, urinary organs (180-181)	0/0.83	0	1/2.79	37
Malignant neoplasms, other and unspecified sites (190-199)	2/3.40	71	15/8.23	137
Leukemia and aleukemia (204)	1/1.25	96	2/2.53	81
Lymphosarcoma, lymphatic and hematopoietic tissues (200-203, 205)	1/2.01	60	5/4.07	126
Diabetes mellitus (260)	2/1.80	134	5/4.56	113
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	28/51.45	65**	125/157.39	81**
Vascular lesions affecting CNS (330-334)	4/5.97	81	9/18.71	49**
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	2/2.39	101	3/4.53	68
Arteriosclerotic heart disease (420)	21/32.54	78*	98/105.39	96
Nonrheumatic endocarditis (421, 422)	0/1.79	0	1/5.35	20
Hypertensive heart disease ([illegible]-443)	1/2.42	49	2/7.01	30
Other hypertensive disease (444-447)	0/0.79	0	3/1.82	170
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/1.55	0	0/2.76	0
Influenza and pneumonia (480-493)	3/2.93	123	2/7.09	29
Ulcer of stomach and duodenum (540,541)	1/1.07	112	1/2.78	37
Appendicitis (550-553)	0/0.24	0	0/0.43	0
Hernia and intestinal obstruction (560, 561, 570)	0/0.42	0	1/1.10	93
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	1/0.40	301	0/0.92	0
Cirrhosis of liver (581)	1/4.49	26	2/11.18	18
Hyperplasia of prostate (610)	0/0.07	0	0/0.33	0
Symptoms, senility and ill-defined conditions (780-795)	1/2.28	53	0/5.09	0
All other diseases (residual)	4/11.97	40	16/26.34	63*
Motor vehicle accidents (810-835)	10/16.14	75	7/16.65	43**
Other accidents (800-802, 840-962)	7/12.94	65*	10/17.82	58*
Suicide (963, 970-979)	6/6.34	114	10/10.28	100
Homicide (964, 980-985)	1/5.80	20	0/6.20	0
Number of workers	2955		4134	
Person-years	34201		43240	

[1] SMR's adjusted for deaths with cause unknown.
*Significant at 5% level.
**Significant at 1% level.

73<

Table 3C

OBSERVED DEATHS/EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS IN VINYL CHLORIDE WORKERS WITH EXPOSURE INDICES BELOW 1.5, BY DURATION OF EXPOSED EMPLOYMENT

Cause of death with I.C.D. number	<60 months exposure obs/exp	SMR[1]	≥60 months exposure obs/exp	SMR[1]
All causes	56/89.23	63**	132/181.28	73**
Tuberculosis (001-019)	0/1.41	0	0/1.97	0
Tuberculosis of respiratory system (001-008)	0/1.31	0	0/1.85	0
Malignant neoplasms (140-205)	8/12.86	73	29/31.46	95
Malignant neoplasms, buccal cavity and pharynx (140-148)	4/0.45	1036	1/1.17	88
Malignant neoplasms, digestive organs and peritoneum (150-159)	1/3.43	34	6/9.08	68
Malignant neoplasms, respiratory system (160-164)	2/3.58	65	9/10.00	93
Malignant neoplasms, genital organs (170-179)	0/0.68	0	2/1.62	127
Malignant neoplasms, urinary organs (180-181)	0/0.55	0	1/1.53	67
Malignant neoplasms, other and unspecified sites (190-199)	1/0.97	120	8/4.43	187
Leukemia and aleukemia (204)	0/0.79	0	1/1.40	73
Lymphosarcoma, lymphatic and hematopoietic tissues (200-203,205)	0/1.26	0	1/2.23	46
Diabetes mellitus (260)	2/1.15	203	3/2.50	124
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	21/33.38	73**	63/86.82	75**
Vascular lesions affecting CNS (330-334)	2/3.93	59	5/10.51	49*
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	2/1.50	155	1/2.49	41
Arteriosclerotic heart disease (420)	16/21.14	88	52/57.87	93
Nonrheumatic endocarditis (421, 422)	0/1.18	0	0/3.02	0
Hypertensive heart disease (440-443)	1/1.58	74	0/3.90	0
Other hypertensive disease (444-447)	0/0.50	0	1/1.02	101
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/0.97	0	0/1.53	0
Influenza and pneumonia (480-493)	3/1.86	188	2/3.95	53
Ulcer of stomach and duodenum (540, 541)	0/0.68	0	1/1.53	67
Appendicitis (550-553)	0/0.15	0	0/0.24	0
Hernia and intestinal obstruction (560, 561, 570)	0/0.27	0	0/0.61	0
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	0/0.26	0	0/0.51	0
Cirrhosis of liver (581)	1/2.80	42	1/6.09	16
Hyperplasia of prostate (610)	0/0.05	0	0/0.20	0
Symptoms, senility and ill-defined conditions (780-795)	0/1.43	0	0/2.79	0
All other diseases (residual)	4/7.45	63	10/12.92	79
Motor vehicle accidents (810-835)	3/9.87	35	5/9.22	56
Other accidents (800-802, 840-962)	3/7.98	44	8/9.85	83
Suicide (963, 970-979)	3/4.08	86	6/5.[illegible]	109
Homicide (964, 980-985)	0/3.55	0	0/3.43	0
Number of workers	1715		2317	
Person-years	21418		23920	

[1] SMR's adjusted for deaths with cause unknown.

*Significant at 5% level.

**Significant at 1% level.

74<

Table 3D

OBSERVED DEATHS/EXPECTED DEATHS AND STANDARDIZED MORTALITY RATIOS IN VINYL CHLORIDE WORKERS WITH EXPOSURE INDICES OF 1.5 OR GREATER, BY DURATION OF EXPOSED EMPLOYMENT

Cause of death with I.C.D. number	<60 months exposure		≥60 months exposure	
	obs/exp	SMR[1]	obs/exp	SMR[1]
All causes	38/47.93	79	119/147.81	81*
Tuberculosis (001-019)	0/0.76	0	0/1.57	0
Tuberculosis of resporatory system (001-008)	0/0.71	0	0/1.48	0
Malignant neoplasms (140-205)	5/6.57	96	36/26.11	141
Malignant neoplasms, buccal cavity and pharynx (140-148)	0/0.23	0	0/0.99	0
Malignant neoplasms, digestive organs and peritoneum (150-159)	1/1.67	76	11/7.47	151
Malignant neoplasms, respiratory system (160-164)	1/1.79	71	12/8.50	144
Malignant neoplasms, genital organs (170-179)	0/0.29	0	1/1.41	73
Malignant neoplasms, urinary organs (180-181)	0/0.26	0	0/1.26	0
Malignant neoplasms, other and unspecified sites (190-199)	1/1.18	107	7/3.51	204
Leukemia and aleukemia (204)	1/0.44	288	1/1.13	90
Lymphosarcoma, lymphatic and hematopoietic tissues (200-203, 205)	1/0.71	178	4/1.84	222
Diabetes mellitus (260)	0/0.61	0	2/2.04	100
Major cardiovascular and renal diseases (330-334, 400-468, 592-594)	7/16.54	54**	62/70.46	90
Vascular lesions affecting CNS (330-334)	2/1.87	135	4/8.19	50
Rheumatic fever & chronic rheumatic heart dis. (400-402, 410-416)	0/0.82	0	2/2.04	100
Arteriosclerotic heart disease (420)	5/10.41	61*	46/47.65	98
Nonrheumatic endocarditis (421, 422)	0/0.57	0	1/2.32	44
Hypertensive heart disease (440-443)	0/0.76	0	2/3.10	66
Other hypertensive disease (444-447)	0/0.27	0	2/0.81	253
Chronic & unspecified nephritis & renal sclerosis (592-594)	0/0.54	0	0/1.23	0
Influenza and pneumonia (480-493)	0/0.99	0	0/3.13	0
Ulcer of stomach and duodenum (540, 541)	1/0.35	362	0/1.25	0
Appendicitis (550-553)	0/0.08	0	0/0.19	0
Hernia and intestinal obstruction (560, 561, 570)	0/0.14	0	1/0.49	209
Gastritis, duodenitis, enteritis and colitis (543, 571, 572)	1/0.14	904	0/0.41	0
Cirrhosis of liver (581)	0/1.56	0	1/5.08	20
Hyperplasia of prostate (610)	0/0.01	0	0/0.13	0
Symptoms, senility and ill-defined conditions (780-795)	1/0.80	158	0/2.30	0
All other diseases (residual)	0/4.02	0	6/11.88	51*
Motor vehicle accidents (810-835)	7/6.05	146	2/7.43	28
Other accidents (800-802, 840-962)	4/4.73	107	2/7.96	26
Suicide (963, 970-979)	3/2.40	158	4/4.62	88
Homicide (964, 980-985)	1/2.18	58	0/2.76	0
Number of workers	1240		1817	
Person-years	12828		19305	

[1] SMR's adjusted for deaths with cause unknown.

*Significant at 5% level.

**Significant at 1% level.

75<

Table 4

SUMMARY OF TOXICOLOGICAL AND EPIDEMIOLOGICAL STUDIES ON VINYL CHLORIDE

HUMAN DATA

				EXPOSURE					
Authors	Species	Sex	No.	Hrs. per Day	Days	Conc. ppm	Total Dose ppm-Days	Observations	Pathology
Von Oettingen (1955)	Human					12,000		Dangerous	
						10,000		Narcosis	
						25,000		Produced symptoms of dizziness, disorientation, headache and burning sensation on soles of feet.	
Gabor Mecca-Radu Manta (1962) Chem. Abstract	Human		82	Workers exposed to DDT, Benzene, Hexachlorocyclohexane, VC, PVC.				Blood: Decrease in catalase Increase in peroxidase, indophenoloxidase and gluthathione Changes occurred during second year of work.	None reported
Lester	Human	M	3	Twice per day		0	0	1/5 slightly dizzy	
Greenberg		F	3	for 3 days,		4,000	83.3	0/6 had any effects	None reported
Adams				5 min. sessions		8,000	166.7	1/6 slightly dizzy	
(1963)				at 6 hours intervals		12,000	250.0	2/6 definitely dizzy	
						16,000	333.2	5/6 dizzy, nausea, blurred vision and heaving symptoms stopped after exposure	
						20,000	416.7	6/6 intoxicated, one with persistent headaches 50% level of no effect is 1.3% No statement about repeated exposures	
Gabor Radu Preda Abrudean Juanof Anca Valczkay (1964) Chem. Abstract	Human		78	PVC Workers				Decrease plasma albumin Increased B and 8 globulin Decrease in B/8 for serum lipoproteins Decrease in serum cholinesterase Decrease in pseudo cholinesterase Normal blood catalase Normal serum pyruvic acid	None reported
Grigorescu Toba (1966) Chem. Abstract	Human			Experimental: PVC Workers Control: Other clinically healthy people				Hypothesis: VC+H_2O>chloral + chloracetic acid (1). Results: (1) was found in 80% of exptl. people, but in none of controls. Most of + findings were in people exposed 2-5 years. In these cases, α-globulin is higher, γ-globulin is lower than people with no (1) in urine, capacity to metabolize (1) decreased after 2 years.	None reported

76<

HUMAN DATA

Authors	Species	Sex	No.	EXPOSURE Hrs. per Day	Days	Conc. ppm	Total Dose ppm-Days	Observations	Pathology
Harris Adams (1967)	Human		2	-	-	-	-		One worker had knee cap and toes involved in the acro-osteolysis. Other worker only hands.
Wilson McCormick Tatum Creech (1967)	Human	M	31	-	-	-	-	No cases of acro-osteolysis diagnosed in 1000 individuals who handled finished resin or used for plastic product production Age range of affected workers 26-47. Incubation period greater than 12 months of polycleaning experience.	31/3000 (3%) workmen associated VC polymerization founds to have acro-osteolysis. 22/31 Acro-osteolysis associated with Reynaud's sumptoms.
Bareeta Stewart Mutchler (1969)	Human	M	13	7.5	1	50	15.6	Breath decay curves, 0 to 20 hrs. after exposure were measured. Level in breath at 4 hrs. is ≃ 1%. No adverse effects noted. About the same set of breath decay curves following occupational exposure.	None Reported
						250	78.1		
						500	156.2		
Kudryautseva (1970) Abstract	Human	M F	50 43	-	-	-	-	1. Changes in ECG: rhythm, conductance, polarization. 2. Increase in systolic index.	None Reported.
Viola (1970) Unpublished	Human	-	1	8	1095			Acro-osteolysis symptoms, Reynaud's syndrome, aversion to fats, enlarged liver	Aerometry: (VC) on factory filters at air discharge time: 2,000 ppm (VC) at point of worker entry: 2,000 ppm in plants where acro-osteolysis occurred-150 ppm (max) in plant with no disease [VC] on gangway and other parts of plant: 10 to 15 ppm.
			5	8	1825			Raynaud's syndrome	
			15		-			Enlarged liver, minor liver insufficiency.	
			500*					13/500 had acro-osteolysis. Olefactory threshold is 0.8 to 1%. Acute nervous symptoms become evident when it is easily perceptible.	

*In several other factories

77<

HUMAN DATA

Authors	Species	Sex	No.	Hrs. per Day	Days	Conc. ppm	Total Dose ppm-Days	Observations	Pathology
Dinman Cook Waterhouse Magnuson Ditcheck (1971)	Human		5011	21,510	Man-years		Experience	Conditions associated with hand cleaning of polymerizers. There appeared to be correlation between reactor degassing time and acro-osteolysis.	25 cases of acro-osteolysis. 16 other individuals questionable Acro-osteolysis appears to be systemic rather than local disease. Reynaud's phenomenon was statistically related to acro-osteolysis.
Dodson Dinman Whitehouse Nasr Magnuson (1971)	Human	M	4	1-23 Months				All patients had worked as PVC reactor-vessel cleaners. Neg. Ca and P balance in one subject. Plethysmographic abnormalities were present in 3 subjects. Esophageal motility within normal limits. Catacholamine, -Hydroxyindole Acetic Acid excretion normal. All other numerous clinical laboratory investigations negative.	Reynaud's phenomenon anteceded osteolytic lesions in all four subjects. ^{18}F scintiscans correlated with radiographic lesions. No liver enlargement or hypothyroidism. Additional smaller abnormalities found in ulnar styloid, oscalcis and patella.
Kramer Mutchler (1971)	Human	M	98	-Up to 25 years Experience				Performed statistical correlation between several clinical measurements and total dose and time-weighted average VC concentration. 2 liver function indices show a positive correlation with total dose (abnormally high). a) Icterus index b) Bromsulphalein 3 other indices are dose-related but are not outside normal limits: a) systolic and diastolic blood pressure. b) hemoglobin negative correlation. c) beta-protein.	None reported.

78<

HUMAN DATA

Authors	Species	Sex	No.	Hrs/Day	Days	EXPOSURE Conc. ppm.	Total Dose ppm-days	OBSERVATIONS	PATHOLOGY
Meyerson Meier (1972) Abstract	Human	-	1						Acroosteolysis Had unique papular skin lesions which have been described only in PVC workers
Markowitz McDonald Fethiere Kerzner (1972) Abstract	Human	-	-						Describes acroosteolysis symptoms. Incidence is <3% among workers
Lange Juhe Stein Veltman (1973)	Human	-	13					Ages 29-52 Reactor cleaners 2-18 yrs. employment	Latent period: 1½-3½ years in 11 patients; 7 and 11 years in other 2 patients. Acro-osteolysis symptoms. Peripheral vessel stenosis. Thrombopeny (low count) is the first objective symptom described in all patients; lung fibrosis orginating in portal system, large spleen, impaired lung function; 10% mortality. This is the first objective symptom described.

HUMAN DATA

				EXPOSURE					
Authors	Species	Sex	No.	Hrs. per Day	Days	Conc. ppm	Total Dose ppm-Days	Observations	Pathology
Marsteller Lelbach Miller Jühe Lange Pohmer Veltman (1973)	Human	-	120	1½ to 21 years.				20 PVC workers were studied out of 45 with suspected skin problems, 30 to 56 years old.	Liver enlarged in 13/20. Pain in Rt. upper abdomen in 2/20. Hyperlipidemia has been diagnosed in 1967 after 6 years in 1/20. Jaundice history in 1955 before exposure in 1968, liver dysfunction was diagnosed, no alcoholism. Spleenomegaly in 7/20. Total bilirubin was 1 mg/100 (which is upper normal limit) in 3/20. Bromsulphalein test was abnormal in 19/20 (>5% retention after 45 minutes). SGOT was >12 mU/ml in 17/20. SGPT was elevated (15-30) in 14/20. Alkaline phosphatase was >48 uU/ml in 2/20 Hypothrombocytemia (<150x 10^3 /mm^3) was found 19/20(<100X10^3/m^3) in 12/20 Acro-ostyeolysis was seen in 4/20. Varicose veins of esophagus in 3/20 Liver histology: Collagen transformation of walls of sinusoids in 5/20. Focal activation of Kupfer cells in 19/20. Focal fatty infiltration in 14/20. Fibrosis of septa and capsule intralobular and portal spaces in 17/20. 15 additional blood parameters were normal. 9 immunological tests were done once, not repeated.

80<

Table 4

SUMMARY OF TOXICOLOGICAL AND EPIDEMIOLOGICAL STUDIES ON VINYL CHLORIDE

ANIMAL DATA

	ANIMALS					EXPOSURE			
Authors	Species	Sex	No.	Hr./day	Days	Conc. ppm	Total Dose ppm-days	Observations	Pathology
Von Oettingen (1955) (Review Article)	Cats	ND	ND	ND	ND	ND	ND	VC is promptly excreted by lungs; 82% is eliminated after inhalation stops	
	Cats	ND	ND	<4	1	100,000 to 130,000	<1,200	Blood VC concentration reaches 15-17 mg%	
	Cats	ND	ND	<4	1	180,000	<30,000	This concentration causes same intra-auricular pressure reduction as 13,000 ppm dichloroethylene, 30,000 ppm ether	
	Cats	ND	ND	<4	1	200,000	<3[illegible],000	Cardiac insufficiency	
	Cats	ND	ND	4	1	250,000 to 300,000	<40,000 to 50,000	Even this does not produce complete cardiac failure	
	Cats	ND	ND	ND	ND	ND	ND	Blood levels are 40 mg% at time of cardiac arrest, 27-30 mg% at time of respiratory arrest	
	Rabbits Dogs	ND	ND	1 min.	1	170,000	118	This is the narcotic concentration	
	Mice	ND	ND	1 min.	1	86,000 to 123,000	60 to 85	This is the narcotic concentration	
	Dogs	ND	ND	<4	1	100,000	<12,000	Cardiac irregularities, ECG abnormalities	
	Dogs	ND	ND	3	7 for several wks	10,000	475	No major change in liver or kidney	
	Dogs	ND	ND	3	7 for several wks	200,000	9,500	Marked salivation, vomiting, respiratory arrest	
	Mice	ND	ND	10 min.	1	245,000 to 295,000	1,700 to 2,040	This is the lethal range for 10 minutes exposure	
	G. Pigs	ND	ND	short time	1	200,000 to 400,000	ND	All killed	

81<

ANIMAL DATA

	ANIMALS					EXPOSURE			
Authors	Species	Sex	No.	Hrs/day	Days	Conc. ppm	Total Dose ppm-days	Observations	Pathology
Von Oettingen (1955) Review Article Continued	G. Pigs	ND	ND	0.5-1	1	100,000	2,000 to 4,000	Dangerous to life	
	G. Pigs	ND	ND	0.5-1	1	5,000	100 to 200	Higher concentrations than this cause severe lung edema and hyperemia of liver and kidney	
	G. Pigs	ND	ND	0.5-1		ND		Order of toxicity is: carbon tetrachloride ≃ chloroform < VC > ethyl chloride	
Mastromatteo Fisher Christie Danziger (1969)	Mice	ND	5	0.5	1	100,000	2,080	Sequential effects were: 1. irritation, 2. increased motor activity, 3. twitching, 4. tremor, incoordination, 5. unconscious, 6. deep narcosis. All animals recovered in 5 minutes.	Mice: Light lung engorgment, kidney swelling; Rats and G. Pigs: same lung picture.
Fisher Christie Danziger (1960)	Rats	ND	5	0.5	1				
	G. Pigs	ND	5	0.5	1				
	Number of animals and duration of exposure same as above					200,000	4,160	1/5 mice died after 30 minutes, same symptoms as above but appeared sooner. Guinea pigs unsteady for 20 min. after exposure.	Lung engorgement, no edema in all species. One rat had fatty liver.
	Number of animals and duration of exposure same as above					300,000	6,250	5/5 mice and 5/5 rats died; 1/5 guinea pigs died; 4/5 guinea pigs recovered in 25 minutes.	Liver and kidney was congested, tracheal epithelium damaged
	Number of animals and duration of exposure same as above					400,000	8,330	2/5 guinea pigs died	Same symptoms, more severe
Torkelson (1) Oyen Rowe (1961)	Rats	M F	10 10	7 7	(5d/wk) (4.5Mo)	500	14,000	Growth and gross appearance were normal. Liver/body weight ratio and absolute liver weight larger than control in males. Liver/body weight and absolute liver weight not larger than control in females Blood SGOT, SGPT, SUN, alkaline phosphatase were normal.	Central lobular liver degeneration. Kidney tubular damage.

ANIMAL DATA

Authors		ANIMALS Species	Sex	No.	Hrs/day	Days	EXPOSURE Conc. ppm	Total Dose ppm-days	Observations	Pathology
Torkelson Oyen Rowe (1961) continued			M F	5 5	7 7	(5d/wk) (4.5mo)	0	0	Control animals	
	(2a)	Rats	M	12	7		200		All groups were normal in appearance, mortality and growth. Hematology (hemoglobin, hematocrit, cells) was normal. Liver function tests (SUN, SGOT, SGPT, alkaline phosphatase) were normal. All organ/body weight ratio normal except M and F rats, where liver/body weight ratio was increased.	Gross pathology was normal. Microscopic pathology was normal in all species except liver of M and F rabbits: Central lobular granular degeneration and necrosis.
			F	12	7	138 exposures in 204 days (6½ months)				
		G. Pigs	M	10	7		200			
			F	8	7					
		Rabbits	M	3	7		200			
			F	3	7					
		Dogs	M	1	7		200	8,050 to 8,400		
			F	1	7					
		Matched controls, both exposed and nonexposed groups				5d/wk				
	(2b)	Same protocols					100	4,000 to 4,200	All animals were normal in appearance, mortality and growth. Hematology (hemoglobin, hematocrit, cells) was normal. Liver function tests (SUN, SGOT, SGPT, alkaline phosphatase) were normal. Liver/body weight ratio of M and F rats were larger than controls.	Gross and microscopic appearance of tissues were normal.

83<

ANIMAL DATA

ANIMALS						EXPOSURE			
Authors		Species	Sex	No.	Hrs/day	days	Conc. ppm	Total Dose ppm-days	Observations
Torkelson	(2c)	Rats	M	5	4	5d/wk	200	4,600	Liver/body/ weight ratio larger than controls, not statistically signifi-
Oyen			M	5	2	for	200	2,300	cant. " " " " " " " " "
Rowe			M	5	1	6.5	200	1,150	Liver/body weight ratio same as controls
(1961)			M	5	0.5	months	200	575	" " " " " " "
continued			M	5	4	as	100	2,300	Liver/body weight ratio higher than controls, not statistically signifi-
			M	5	2	above	100	1,150	" cant. " " " " " " " " "
			M	5	1		100	575	Normal in all respects
			M	5	0.5		100	265	" " " "
	(3)	Rats	M	24	7				All parameters normal in all species.
			F	24	7	130			
		G. Pigs	M	12	7	exposure			
			F	12	7	in			
		Rabbits	M	1	7	189			
			F	3	7	days			
		Dogs	M	1	7				
			F	1	7				
		Matched controls, exposed and unexposed groups.							
Lester		Sherman	ND	2	0-2	1	50,000	0-4,160	Moderate intoxication, righting reflex lost.
Greenberg		rats	ND	2	0-2	1	60,000	0-5,000	More intense intoxication, righting reflex present.
Adams	(1)		ND	2	0-2	1	70,000	0-5,830	More intense intoxication, righting reflex lost.
(1963)			ND	2	0-2	1	100,000	0-8,330	Corneal reflex disappears, no gross pathology.
			ND	1	5 min.	1	150,000	552	Deep anesthesia.
					42 min.	1		4,380	Respiratory failure of same animal.
			ND	1	2	1	150,000	12,500	Deep anesthesia, complete recovery after exposure. No pathology observed.

ANIMAL DATA

85<

Authors		Species	Sex	No.	EXPOSURE Hrs. per Day	Days	Conc. ppm	Total Dose ppm-Days	Observations	Pathology
Lester Greenberg Adams (1963) Cont'd	(2)	Sherman rats	M	9	8	2 then 13	100,000 80,000	Variable	After animal deaths, replacements were made in chambers. Two males survived all 15 exposures. Remaining animals and replacements survived an average of eight exposures	
			F	9	8	Same exposures as above.			One died after two exposures at 100,000 and twelve at 80,000. One died after two exposures at 100,000 and twelve at 80,000. Six/nine survived all fifteen exposures.	
			M	9	8		0		Control animals	
			F	9	8		0		Control animals	
									Growth stopped during exposures and resumed at normal rate after exposures. External appearance normal. Liver color, appearance, consistency, degree of congestion was same as controls.	Lungs had focal pneumonia which healed after two weeks of recovery from exposure. One-third of animals had parasitic cysts in liver. Liver pathology same as controls, but more variation in amount of fatty infiltration. Spleen had advanced lymphocytic hyperplasia. Kidney pathology same as controls.
	(3)	Sherman rats	M	15	8	5days/week for 3 months	20,000	434,000		
			F	15	8		20,000	434,000	1/30 died. External appearance of all animals normal. Liver larger, spleen smaller than controls. White blood cells lower, lymphocytes higher, neutrophils lower than controls. Body weight and hemoglobin were same as controls.	All organs had normal gross appearance. Liver parasitic cysts in all animals. Liver fat normal. No abnormal histology. Congestion and swelling greater in liver than controls.
			M	15	8	Same	0	0	Control animals.	
			F	15	8	Same	0	0	4/30 died.	Congestion and swelling less in kidney than controls. Congestion and swelling same in spleen as controls.

ANIMAL DATA

Authors		Species	Sex	No.	EXPOSURE Hrs. per Day	Days	Conc. ppm	Total Dose ppm-Days	Observation	Pathology
Lester Greenberg Adams (1963) Cont'd	(4)	*Sherman* rats	M	5	8	19	50,000	317,000	*No mortality.* On days 1-4, animals lost weight, showed neuorological symptoms. On days 4-19, weight gain was normal. Serum transaminase, hematocrit, and prothrombin times were normal. White and red cell counts were lower than controls. Hair: all 5 males had thin hair and scaly tails; females and controls were normal. Liver/body weight ratio was higher than controls.	*Gross organ appearance was* same as controls. Liver pathology showed congested cells. Liver parasitic cysts seen in all animals.
			F	5	8	19	50,000	317,000		
			M	5	8	19	0	0	Control animals.	
			F	5	8	19	0	0	Control animals.	

ANIMAL DATA

Authors	Species	Sex	No.	EXPOSURE Hrs. per Day	Days	Conc. ppm	Total Dose ppm-Day	Observations	Pathology
Kuebler (1964) Abstract	Rats Mice G. Pigs	ND ND ND	ND ND ND	2 2 2	100 100 100	5,000 15,000 50,000	41,600 125,000 416,000	No effect at 5,000 and 150,000 ppm. At 50,000, animals were hyperactive, but returned to normal after exposure.	No histological damage
	Mice	ND	ND	0.5 minutes		Distance 20-25cm.	ND	Animals sprayed with a shellac-based hair spray	No change in lung histology
Vazin Plokhova (1968a) Abstract	Rabbits	ND	ND	"chronic"		3,500 3,900	ND	Brain electrical activity changes: Appearance of beta waves (80 Hertz) in anterior and posterior hypothalamus along with circulatory changes.	
Vazin Plokhova (1968b) Abstract	Rabbits	ND	ND	4	167 (5.5 mos.)	3,500 to 3,900	ND	Decreased heart rate, arrhythmia Decreased ECG voltage Decreased duration of systole Reduced blood flow. Increased arterial pressure.	Altered β waves in EEG from posterior hypothalamus. Potentials from anterior and posterior hypothalamus increased by 18-30% and 70-85% respectively.
Vazin Plokhova (1969a) Abstract	Chinchilla rabbits	ND	8	4	150	8 to 12	200 to 300	After 20 days, blood adrenaline rose from 3.5 μgm% to 6.15 μgm%; at 40 and more days, it was 6.6 μgm%. Posterior hypothalamus electrical activity also changed. This is the direct cause of hypertension.	
Vazin Plokhova (1969b) Abstract	Rats	ND	ND		150 (5 months)			Disrupted cardiac work rhythm. Bradycardia and arrhythmia. Reduced relative duration of I-II and T-II intervals. Relative duration of QRS complex did not change. After 15 days recovery: cardiac activity rhythm returned to normal, but the duration of the sound interval remained below initial levels for another 15 days. Therefore, max. permissible VC concentration is significantly less than .03mg/1 (12ppm).	

87<

ANIMAL DATA

				EXPOSURE					
Authors	Species	Sex	No.	Hrs. per Day	Days	Conc. ppm	Total Dose ppm-Days	Observations	Pathology
Clapp Kaye Young (1969) Abstract	Rats	ND	ND	-	1	Sub-Cutaneous	ND	Urine contains allylmercapturic acid and 3-hydroxypropylmercapturic acid. These compounds arise by the reactions of allyl compounds with glutathiones.	
Viola (1970a)	Wister Rats	M M	25 25	4	260 (5days per wk for 12 months)	30,000 0	$1,300x10^3$	Animals slightly sleepy during exposure. Gross behavior deteriorated after 10 months. 13/50 died of cardio-respiratory complications. 2/50 died of bleeding in the peritoneal cavity. No mention of skin tumors.	Most animals had pathological involvement of brain, liver, kidney, thyroid. Severe proliferation of cartilege and bone abnormalities in small metatarsal bones. Severe tissue degeneration in brain and liver and thyroid. Connective tissue invaded small arteries in feet. Enlarged, proliferating Kupfer cells in liver.
Viola (1970b)	Rats Wister 300 gm	-	90	1	1	10,000	417	Distribution of VC in tissue: Red cells had much more VC than serum-high variation VC is in urine, but major quantity is lost via lungs. (VC) falls rapidly in first hour in expired air, blood, urine, and brain, liver kidney. After 3 hrs. no VC is measurable.	None observed.
Viola Bigotti Caputo (1971)	Rats Wister	M	26	4 5day/wk	260	30,000	$1300x10^3$		Controls showed no tumors. Almost all exptl. animals developed skin and lung tumors. Very few bone tumors; when seen they were in all 4 extremities. 65%-70% of tumors were skin tumors near parotid and submaxillary glands. Frequencies: SKIN LUNGS BONE 26/26 16/26 16/26 Lung tumors were glandular. New cartilage and subsequent ossification in 4 extremeties. Hard mass first seen after 10 months exposure.
		M	25	1 5day/wk	260	0	0		

88<

ANIMAL DATA

				EXPOSURE					
Authors	Species	Sex	No.	Hrs. per Day	Days	Conc. ppm	Total Dose ppm-Days	Observations	Pathology
Basalaev Vazin Kochetkov (1972) Abstract	Rabbits Rats	ND ND	ND ND	ND ND	6 mos. (130 to 180 days)	12-16	ND	Changes in electrical activity of hypothalamus Hyperadrenalinemia. Cardio-vascular function impaired. Bone resorption and osteoporosis. Theory: All symptoms are caused by hypothalamus disfunction and subsequent hormone imbalance.	

ANIMAL DATA

Authors	Animals			Exposures				Observations		Pathology		
	Species	Sex	No.	Hrs/Day	Days	conc. ppm	Total dose ppm-days	Survivors	Total	Liver Angiosarcomas	Zymbal Sarcomas	Nephro-Blastomas
Maltoni 1974	Rats Sprague-Dawley	M F	309 268	4 5da/wk	635	10,000	$<1060X10^3$	0/69	27	6	13	3
						6,000	$<635X10^3$	0/72	21	11	5	3
						2,500	$<265X10^3$	0/74	21	9	2	6
						500	$<52.9X10^3$	0/67	16	7	3	3
						250	$<26.5X10^3$	1/67	11	2	0	5
						50	$<5.3X10^3$	3/64	0	0	0	0
						0	0	1/68	0	0	0	0
	Rats Sprague-Dawley	M F	265 280	4 5da/wk	280	10,000	$466X10^3$	36/60	3	0	3	0
						6,000	$280X10^3$	43/60	1	0	1	0
						2,500	$167X10^3$	54/60	0	0	0	0
						500	$23X10^3$	56/60	0	0	0	0
						250	$11.7X10^3$	44/60	0	0	0	0
						50	$2.3X10^3$	50/60	0	0	0	0
						0	0	183/190	0	0	0	0
	Rats Sprague-Dawley	M F	30 30	4 5da/wk	155	30,000	$775X10^3$	60/60	2	0	2	0
	Rats breeders	M F	36 110	4 (day 12-18 of preg.)	7	10,000	11667	28/30	0			
						6,000	7000	28/30	0			
	off-spring					10,000	11667	30/34 (?)	1 (subcutaneous angiosarcoma)			
						6,000	7000	30/32	1 (subcutaneous antiosarcoma)			

90<

ANIMAL DATA

Authors	Animals			Exposure			Observations*	Pathology
	Species	Sex	No	hrs/day	days	conc. ppm	Survivors	liver angiosarcomas
Industrial Bio - test Laboratories	mice Swiss CD-1 mice	M	300	7 5da/wk	165	2500		
						200	143/200	17
						50		
							166/200	4
		F	300	7 5da/wk	165	2500		
						200	157/200	2
						50		
	rats Sprague Dawley outbred COBs	M	300	7 5da/wk	165	2500 200 50		
		F	300	7 5da/wk	165	2500 200 50		
	hamster Golden Syrian	M	300	7 5da/wk	165	2500 200 50	*as of April 15, 1974	
		F	300	7 5da/wk	165	2500 200 50		

91<

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94<

APPENDIX VIII

DISPOSAL OF PRODUCTS CONTAINING POLYVINYL CHLORIDE

This discussion on disposal of PVC emphasizes incineration and landfilling, the only presently used large-scale methods for the disposal of solid wastes. There is also a limited discussion of resource recovery possibilities.

Incineration

The two areas of concern related to PVC incineration are incinerator air pollution and incinerator and gas scrubber corrosion.

Hydrogen chloride is the major toxic material released when PVC is burned. It has been shown that virtually all of the chlorine is released from PVC on combustion, resulting in HCl. It is estimated that 0.2 percent of solid waste is PVC, and 16×10^6 tons per year of solid waste are incinerated in the United States. Thus, on the order of 32,000 tons of PVC are burned annually, releasing approximately 18,500 tons per year of HCl as air emissions.

Other solid waste sources which can produce HCl are chlorides in food waste, plants, grass clippings, and inorganic salts. The formation of compounds requires volatilization and reaction with incinerator flue gases. Achinger and Baker compiled data indicating an emission factor of six pounds of HCl per ton of solid waste burned. Recent data on HCl emissions obtained by Battelle show a factor of 5.1 pounds per ton. A value of five to six pounds per ton would be a reasonable emission factor to use for HCl emissions from municipal incinerators. Using an emission factor of 5.5 pounds per ton gives 44,000 tons per year of HCl produced by incineration of municipal solid waste. The amount of HCl produced from PVC using the above calculation is 42 percent of the total.

Much more HCl is probably now emitted to the atmosphere from the nation's coal-burning power plants than from our municipal incinerators. However, there still could be a hazard in the immediate vicinity of an incinerator as a direct result of its HCl emissions. Of particular concern is the possible dispersal of the stack gases to cause the ambient concentrations of HCl at ground level to exceed harmful concentrations. However, HCl is not at the present time regulated by EPA.

Other air pollutants could be formed from the additives in PVC during incineration. Several additives are usually incorporated into the polymer to emphasize particular properties not inherent in the base polymer. The types of additives are antioxidants, antistatics, colorants, fillers, plasticizers, and stabilizers. Some of the additive agents used are: antioxidants--phenols, amines, phosphates, and sulfur compounds; antistatics --amine derivatives, quaternary ammonium salts, phosphate esters, polyethylene glycolesters; colorants--salts or oxides of metals, aluminum, copper and inorganic pigments; fillers--silica, glass, calcium carbonate, metallic oxides, carbon, cellulose fillers, asbestos; plasticizers--phthalates, organic phosphates; stabilizers--lead salts of acids, barium, cadmium, calcium, zinc, alkyl tin compounds.

It is highly unlikely that large quantities of VC will be emitted during incineration of PVC. There is no evidence that PVC will chemically revert to VC. Some small amounts of entrapped monomer might conceivably survive incineration, but these quantities would be very low.

The second area of concern with incineration of PVC is firebox corrosion and corrosion of pollution control equipment. HCl can be a major factor related to corrosion of this equipment during incineration at certain temperatures. In the case of plastics, PVC is the major source of chlorine leading to HCl, but other plastics may also contain some chlorine. Incinerators with heat exchangers will have corrosion problems on the fire side of the exchange equipment when the combustion gases contact the outer metal surface. Other surfaces of concern are in the cooling area and in the gas scrubbers.

Estimates indicate that in incinerators with heat-recovery systems PVC in the refuse will increase tube maintenance costs by 15 to 20 perpercent over that to be expected if PVC-free refuse was used as fuel.

About 95 percent of the incinerators in this country have some type of air pollution control equipment that is exposed to the high chloride environment resulting from refuse combustion. Because of the high chlorine content of the combustion products, the cooling and precipitating water from the scrubbers that contacts the flue gas contains large quantities of chloride and is extremely corrosive to the structure.

In summary, technology exists for controlling the HCl emissions that result from incineration of solid waste; however, the application of this technology will result in increased costs. If technology is not applied, then the contribution of PVC to the nation's air pollution problem will increase because of the projected increases in the usage and disposal. HCl scrubbing technology is available, but its application results in corrosion problems. Depending on construction materials, design, and operation, these problems can be either large or small.

Landfilling

PVC does not decompose significantly within the normal time frame of most other municipal solid wastes. It comprises only about 0.2 percent of the total municipal solid waste being landfilled today, and the effect of PVC on the reuse of the landfill site, at least in the short run, should be negligible.

Since PVC degrades very slowly, in the landfill environment it should not add significantly to the production of leachate or decomposition gases as do other parts of the refuse. The additives of greatest concern are probably the plasticizers. However, if a sanitary landfill is designed and operated with today's technology, disposal of PVC products in a sanitary landfill should pose no special problems to the operation or to the ultimate use of the site.

Resource Recovery

Recycling of solid waste is a growing industry. Technology has been developed to recover some resources from many of the items in the municipal waste stream. However, the technology to separate plastics or PVC from the waste stream has not yet been commercially demonstrated. The solution to the separation of plastic waste from other components of the municipal waste stream is one deterrent to direct recycling and reuse of plastics, including PVC. However, gathering and centralizing the waste products are also major problems.

Some types of scrap PVC from the fabrication process are presently being recycled back into the manufacturing process. This reduces the solid waste from plastic fabrication plants and reduces the need for new raw materials.

There is work underway to develop means for utilizing the benefits of recycling the total municipal waste stream. Examples of these recycling techniques are listed below:

-- To recover heat given off during the incineration of solid waste containing PVC and other combustible materials as electricity or steam for heating. An example is EPA's research contract with the Combustion Power Company of Menlo Park, California, in which combustion gases are expanded through a turbine to produce power.

-- To recover the products of a refuse pyrolysis operation either as a pipeline gas or as feed material for a nearby refinery. An example is EPA's research grant with West Virginia University in which refuse pyrolysis is being studied on a bench-scale. A second example is the Bureau of Mine's research effort to convert refuse to pipeline gas. Also, US and Japanese industrial firms are actively exploring this area.

The recent change in the world's supply of crude oil should speed up research and development on new and existing ways to utilize more fully the resource of waste PVC.

97<

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ACTIVITIES OF TASK FORCE

The principal activities undertaken or stimulated by the Task Force are set forth below:

MARCH - Recognition of problem of pesticidal sprays containing VC -- Responsibility assigned to Office of Pesticide Programs

MARCH - Analysis of material losses during PVC polymerization process

MARCH 19-21 - Pilot monitoring effort at B. F. Goodrich Plant in Louisville

MARCH - Preliminary evaluation of health effects data

APRIL 2 - Meeting with representatives of PVC manufacturers organized by Manufacturing Chemists Association

APRIL 4 - Meeting with representatives of interested environmental groups

APRIL - Development of interim methodology for VC sampling and analysis

APRIL/MAY - Visits to VC manufacturing facilities and to PVC polymerization, compounding, and fabrication facilities

APRIL 12 - First of series of interagency meetings convened by EPA

APRIL/MAY - Monitoring at seven complexes involving 10 PVC and 2 VC plants

APRIL/MAY - Review of health effects data

APRIL 30 - Review of Industrial Biotest toxicological experiments

MAY 27-31 - Preliminary VC water persistence studies

MAY - Preliminary VC air persistence studies

MAY/JUNE - Recognition of air emissions problem -- Responsibility assigned to Office of Air Quality Planning and Standards

JUNE 3 - Technical review of monitoring activities

JUNE 11 - Administrator's meeting with senior executives of 29 companies producing PVC and VC

JULY - Development of improved methodology for VC sampling and analysis